DISSERTATION

A CROSS SECTIONAL STUDY ON

"THE EFFECT OF WHOLE BODY VIBRATION WITH NOISE ON HEARING IN DRIVERS AND CONDUCTORS".

M.S DEGREE EXAMINATION

BRANCH IV

ENT AND HEAD & NECK SURGERY

This Dissertation is submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment of the University Regulations for the award

of

Degree of MASTER OF SURGERY (OTORHINOLARYNGOLOGY)

Thanjavur Medical College, Thanjavur

2016

CERTIFICATE

I certify that the Dissertation titled "A CROSS SECTIONAL STUDY ON THE EFFECT OF WHOLE BODY VIBRATION WITH NOISE ON HEARING IN DRIVERS AND CONDUCTORS" submitted by Dr. PALANIVEL P., for Degree of Master of Surgery (Otorhinolaryngology) to The Tamilnadu Dr.M.G.R. Medical University, Chennai is the result of original research work undertaken by him in the department of ENT AND HEAD & NECK SURGERY, Thanjavur Medical College, Thanjavur.

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DECLARATION

I hereby declare that the dissertation titled**, "A CROSS SECTIONAL STUDY ON THE EFFECT OF WHOLE BODY VIBRATION WITH NOISE ON HEARING IN DRIVERS AND CONDUCTORS"** a clinical study submitted by me is a result of original work carried out by myself under the guidance of **Prof. Dr .RAVINDRAN, MS, DLO., ASSOCIATE PROFESSOR and retired chief of Dept. of Otorhinolaryngology, Thanjavur medical college,** Thanjavur and with present guide **Prof. Dr. G. GANDHI, M.S., D.L.O., professor and chief of Dep. of otorhinolaryngology, Thanjavur medical college, thanjavur.** This is submitted to **THE TAMILNADU DR. M G R MEDICAL UNIVERSITY, CHENNAI**, in partial fulfilment of the requirement for the degree of **MASTER OF SURGERY, (ENT, HEAD & NECK SURGERY).**

PLACE: Dr. Palanivel P

Date:

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This is to certify that The Research Proposal / Project titled

A CROSS SECTIONAL STUDY ON WHOLE BODY VIBRATIONS WITH NOISE ON HEARING IN BUS DRIVERS AND CONDUCTERS

submitted by Dr. P. PALANIVEL **of**

Dept. of ENT **Thanjavur Medical College, Thanjavur**

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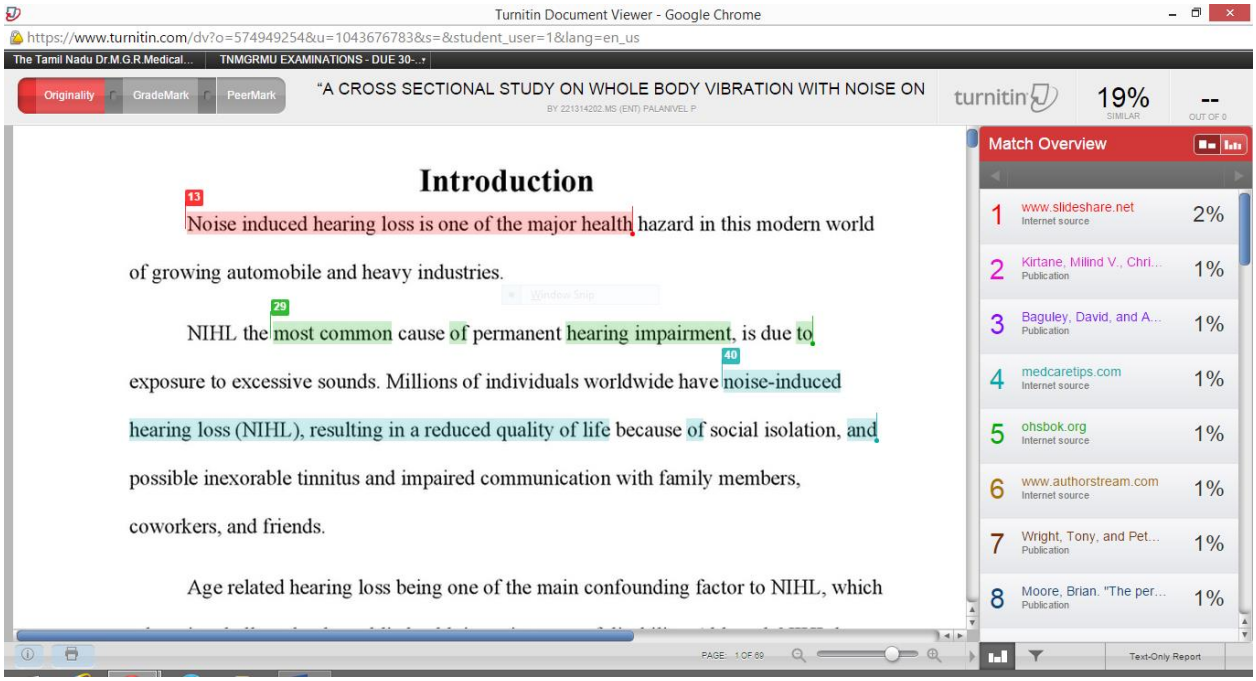
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Introduction
Noise induced hearing loss is one of the major health hazard in this modern world of growing automobile and heavy industries.
NIHL the most common cause of permanent hearing impairment, is due to exposure to excessive sounds. Millions of individuals worldwide have noise-induced hearing loss (NIHL), resulting in a reduced quality of life because of social isolation, and possible inexorable tinnitus and impaired communication with family members, coworkers, and friends.
Age related hearing loss being one of the main confounding factor to NIHL, which
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Introduction

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NIHL the most common cause of permanent hearing impairment is due to exposure to excessive sounds. Millions of individuals worldwide have noise-induced hearing loss (NIHL), resulting in a reduced quality of life because of social isolation, and possible unresolvable tensions and impaired communication with family members, coworkers, and friends.

Age-related hearing loss being one of the main confounding factor to NIHL, which otherwise shall not be the public health issue in terms of disability. Although NIHL has been studied experimentally for more than a century, only in the last few decades we have some major breakthrough occurred in our basic understanding of the ear's reaction to damaging sounds with a better understanding of the environmental and genetic factors that contribute to NIHL.

Although all people are subjected to noise exposure, drivers and conductors are the ones exposed for longer duration where personal protection could not be emphasized other than modifying their working environmental factors. Ultimately noise is produced by vibration of some particle by which sound propagates, but depends on impedance of media through which it travels. Naturally metals are known for its vibration conduction, where drivers are exposed in direct contact but conductors are not so. ISO 1999 states a damage risk threshold equal to 75-85 hz noise produced by our commercial transport

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Contents

Abbreviations

TTS: Temporary threshold shift.

PTS : Permanent threshold shift.

NIPTS: Noise induced permanent threshold shift.

NIHL: Noise induced hearing loss.

WBV: Whole body vibration.

HAV: Hand arm vibration.

VWF: Vibration induced white finger.

dB: decibel.

SPL. : sound pressure level

GDPF: Glial cell derived neuro protector.

ROA: Reducing oxidation agents.

HFL : High frequency loss.

LFL: Low frequency loss.

MCL: Master calculator loss of hearing impairment after eliminating age factor %.

KHZ: kilo hertz.

SNHL: sensorineural hearing loss

AC:air conduction.

BCL: bone conduction loss.

OAE: otoaccoustic emission.

DPOAE: distorted potential otoaccoustic emission.

D: driver.

C: conductor.

Introduction

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Although all people are subjected to noise exposure, drivers and conductors are the one exposed for longer duration where personal protection could not be emphasized other than modifying their working environmental factors. Ultimately noise is produced by vibration of some particle by which sound propagates, but depends on impedance of media through which it travels. Naturally metals are known for its vibration conduction, where drivers are exposed in direct contact but conductors are not so. ISO 1999 states a damage risk threshold equal to75 dB but noise produced by

our commercial transport buses were measure to 80-92 dB and vibration touches to a maximum 4.0 m^2in driver cabin for front engine loaded vehicles.

Vibration is the basic behavior by which sound travels, but vibration could not be isolated without minimal sound. Drivers are exposed to both noise and vibration at the same time, but conductors, who always accompany with drivers does not experience vibration rather noise similar to drivers. Drivers and conductors of the commercial transport are subjected to noise and vibration at the same time and for longer duration. So by screening them we can study the synergistic effect of vibration with noise in NIHL.

Though NIHL in occupational disease depends on many factors we feel vibration could be the main apart from noise that causes health hazard physically and psychologically. Effect of Vibration as a lone factor has been studied in experimental model and proved no contribution on hearing loss. But such an environment in reality without noise effect is not possible for drivers where hearing could not be attenuated. Very few studies have been documented for synergistic effect of vibration and noise for NIHL (NIPTS) in our literature.

So in this study we prefer to evaluate the impact of vibration on NIHL indirectly by comparing clinically healthy drivers and conductors, who are referred to medical health checkup from state transport corporation.

OBJECTIVES

- To study the synergistic effect of vibration and noise on SNHL in bus drivers and conductors by comparison.
- To study the present hearing status of bus drivers and conductors.
- To study noise effect on drivers and conductors for Prevention of occupational hearing health hazard in drivers and conductors.
- To insist on implementation and the importance of "Noise and vibration management program".
- To improve driver's performance and safety measures for passengers by insisting on vehicle cabin ergonomics refinement and improving roads.

Review of history and literature

3.1 Definition;

Noise is defined as non-harmonic sound that is harmful to health and safety of humans.

Vibration is defined as oscillatory motion of the solid that arises from mechanical status which is in contact with humans.

Literature:

Occupational noise

- Effect of noise on humans has been recognized throughout history. The earliest notation is attributable to Pliny the Elders 23-89AD naturalis historiae, refers to the environmental noise of the Nile falls and its ill effect on local inhabitants (NIOSH, 1998; Rosen, 1974)
- In ancient period carts were banned in night hours in residential area due to its illeffect.
- Ramazzini (1633-1714) described the hearing loss of coppersmiths in De Morbis Artificum Diatriba (occupational disease).
- Due to industrial revolution NIHL were increased in railway workers, was studied first by Goostein and Kayser (1881) on Germans. In 1886 Thomas Barr reported on hearing impairment in Scottish boil makers.
- Georg von Bekesy discovered the `travelling wave' by which sound is propagated in air and communicated with cochlea, received noble prize in 1961.

- In 1970,Burns and Robinson proposed `the concept of admission' which is based on equal energy hypothesis to describe the energy lost by workers due to noise exposure.(NIOSH,1998).this states ``equal amount of energy will produce equal hearing loss, regardless how the sound energy is distributed in time'' which formed the basis for Occupational Safety and Health recommendation for a 3-dB exchange (for a 3-dB increase in noise level the exposure time must be halved to balance energy exchange concept. Modern noise regulation standards uses the above concept ISO, 1990 and 1999.
- In 1862 Dr Maurice Raynaud, first described the vibrational effect on humans by describing Raynaud's syndrome, followed by Professor Giovani Loriga described the syndrome in miners. In 1918 the first comprehensive study of vibrating tools was done by Dr Alice Hamilton. (A study of spastic anemia in stone cutters).
- Robinson and Sutton played a major part in identifying age related hearing loss in the field of NIHL for OSHO standards (68).
- A more objective system of grading the "Effects of vibration induced Reynaud's syndrome and neurological effect" was proposed and accepted at work shop on "hand arm vibration syndrome" (e.g. Griffin, 2006; IIAC, 1995).
- Synergistic effect of noise and vibration on hearing was studied in animal and experimental model, but there is no evidence based large scale study which is
- still infantile. Griffin in his Hand book of vibration, 2006 mentioned about the SNHL in synergistic effect of noise and vibration but no effect on isolated vibration exposure in experimental model.

Occupational hearing loss one of the most common professional disease progresses over years of noise exposure in work related environment causing damage to hearing without the knowledge of subject, which is irreversible and untreatable but can be prevented. Many factors can be involved in hearing loss that necessitates the further refinement in heavy engineering occupational profession.so here I prefer impact of vibration on hearing loss in drivers and conductors of the transport corporation who are subjected to noise with vibration for a long period in their life. There seems no enough studies for the impact of hand foot and body vibrations on hearing.

Since there is no medical treatment for this type of hearing loss, it is evident the importance of preventive and conferences aimed at preserving hearing

To carryout study on vibration and noise effect on hearing we need enough knowledge about anatomy, physiology and pathology of an auditory system.

4.2 Anatomy of an auditory system.

Auditory system is comprised of external, middle and inner ear with neural pathway.

4.2.1 External ear:

Comprises auricle (pinna) EAC and tympanic membrane. The pinna of humans is composed mostly of cartilage and has no useful muscles and is continues with EAM meatus. The center of the pinna, the concha, leads to the external auditory meatus, which is about 2.5 cm long. The lateral third of the canal is the cartilaginous portion. It contains cerumen-producing glands and hair follicles. The remaining medial two thirds is the bony portion, including an epithelial lining over the tympanic membrane

Whereas bony part lined is by the thin immobile skin which lacks hair and gland, is continuous with TM. The lateral Bony canal forms isthmus, the narrowest part.

Blood supply; posterior auricular A and superficial temporal A branch of external carotid A.

Tympanic membrane:

The TM forms the medial wall of EAC and lateral wall of middle ear and form 55°with floor laterally. Composed of four layers outer epithelial middle fibrous (radial and circumferential) and medial mucosal. It is concave shaped membrane connected centrally to the manubrium of malleus and peripherally to tympanic sulcus. The tip of the malleus is attached to the depression known as umbo. Both the fibrous layers become integrated peripherally to form annular ligament which rests in tympanic sulcus. This tympanic sulcus terminates superiorly at anterior and posterior spines, to which the most superior edge of the fibrous layer is attached, which form posterior

and anterior malleolar folds that insert on the lateral process of the malleus. The small area above anterior and posterior malleolar folds is called pars flaccida or shrapnel's membrane and thicker inferior area is known as pars tensa.

Arterial supply of TM arises from both external auditory meatus and middle ear by anterior tympanic branch of maxillary artery, the stylomastoid branch of the osterier auricular artery and the middle meningeal artery. Nerve supply to TM are branches of auriculotemporal nerveV3 auricular branch of vagus and tympanic branch of glossopharyngeal nerve.

4.2.2 The tympanic cavity (middle ear)

The middle ear cavity can be spatially divided into hypotympanum,

Mesotympanum, and epitympanum, the limits of which are defined by their location relative to the tympanic anulus. The mesotympanum is the space just medial to the tympanic membrane, which extends from the Eustachian tube opening anteriorly to the facial nerve posteriorly. The epitympanum or attic, lies above the level of malleolar folds and is separated from meso and hypotymanum by series of mucosal folds. The hypotympanum lies below the level of tympanic membrane and is continuous with mesotympanum. Lateral wall tympanic cavity is formed by bony lateral wall of epitympanum (scutum) superiorly, tympanic membrane centrally and bony lateral wall of hypotympanum inferierly. Two canal and a fissure can be identified in the lateral wall, petrotympanic fissure transmits anterior tympanic artery, canal of hugier and posterior canaliculus transmits chordae tympanie anteriorly and posteriorly. Roof is formed by tegmen tympani a bony plate that separates middle ear from middle cranial fossa. Veins from tympanic cavity pass through petrosquamous suture line to superior

petrosal sinus. Floor seperates middlr ear from jugular bulb and at the junction of the floor and medial wall there is a small hole transmits glossopharyngeal nerve.

The anterior wall perforated by superior and inferior carotico-tympanic nerve carrying sympathetic branch to tympanic plexus. The middle third comprises the tympanic orifice of Eustachian tube (5*2mm) just above it tensor tympani canal .the upper third pneumatised houses anterior sinus.

Major part of the medial wall formed by promontorium part of the basal coil of cochlea and usually has grooves for tympanic plexus. Behind and above is the oval window (3.25L and1.75mm wide), which connects cavity with vestibule, but closed by foot plate of stapes. The round window (2.3*1.9) niche lies below and little behind the oval window, which is separated by posterior extension of promontorium called subiculum. Occasionally another ridge of bone extend from promontorium superiorly above subiculum to pyramid called ponticulus. Facial canal runs above the promontory in between oval window and lateral semicircular canal in anterio-posterier direction and posterior to processes cochleariformis in the medial wall of epitympanum. In front of it swelling corresponds to geniculate ganglian.

The Posterior wall is wider above than below and has an opening called additus ad antrum that leads to posterior epitympanum and mastoid antrum. Below it forms fossa incuda houses incudalis and medial to opening of chordae conical pyramid that gives attachment to stapedes tendon. Lateral to pyramid is the facial recess. Medial to pyramid mesotympanum extends to form sinus tympani.

Contents of the tympanic cavity:

The tympanic cavity contains ossicles, two muscles, the chorda tympani and tympanic plexus. The ossicles are the malleus, incus and stapes that form a semi rigid bony chain for conducting sound.

Malleus:

The malleus is the largest of the three, measuring up to 9mm in length.it comprises a head, neck and handle or manubrium. The head lies in the epitypanum and suspended by a superior ligament.it has a saddle shaped medial facet to articulate with incus. Below the neck the bone broadens and give rise to the lateral process, the anterior process and the handle. The lateral process is the prominent landmark in tympanic membrane and receives the anterior and posterior malleolar folds from the tympanic annulus. Chorda tympani crosses above this point medially but below the neck. A slender anterior ligament arises from the anterior processes to insert into the petro-tympanic fissure. The handle runs downwards between the mucosal and fibrous layer of tympanic membrane and forms umbo at the mid-point tenting medially.

The incus:

The incus articulates with the malleus and has a body, short and process. The body lies in the epitympanum and articulates with the malleus. The body of the incus is suspended by the superior incudal ligament attached to the tegman tympani. The long process descends into the mesotymanum behind and medial to malleus form a slender lenticular recess that articulates with the stapes.

The stapes:

The stapes is shaped like a stirrup and consist of a head, neck, the anterior crura, the posterior crura and a foot plate. The head faces laterally and has a cartilage facet that articulate with the incus. The stapedius tendon inserts into the neck and posterior part of the posterior crura. Both anterior and posterior crura are hallowed out on their concave surfaces, which gives an optimum strength and lightness. The two crura joins the footplate, which always has convex superior margin and flat inferior. The average dimension of the footplate are 3mm long and 1.4mm wide, and it lies in the oval window where it is attached to bony window by annular ligament.

The stapedius muscle:

The stapedius arises from the walls of the conical cavity with in the pyramid and form the downward curved continuation of this canal in front of the facial nerve. A slender tendon arises from the apex of the pyramid and inserts into the stapes. Nerve supply by facial nerve.

The tensor tympani muscle:

This is the long slender muscle arising from the walls of the bony canal lying above the Eustachian tube. From its origin the muscle passes backwards into the tympanic cavity where it lies on the medial wall a little below the facial nerve. This enters the processes cochleariformis where it is held down by a transverse tendon as it runs through a right angle to pass laterally and insert into medial aspect of the upper end of the malleus handle. Nerve supply by the medial pterygoid branch of mandibular nerve.

The tympanic plexus:

Plexus formed by the tympanic branch of glossopharyngeal nerve (Jacobson's nerve) and carotico tympanic branches from internal carotid sympathetic plexus.

Mucosa of tympanic cavity:

Lined by mucus secreting respiratory mucosa bearing cilia on its surface. Three distinct mucociliary pathways can be identified- epitympanum, promontorial and the hypotympanum. Each pathways coalesces at tympanic orifice of the Eustachian tube. The mucosal folds covers all the structures as like peritoneum and forms compartments that carries blood vessels. Prusack's space, formed by pars flaccida laterally and the neck of the malleus medial, bounded by lateral malleolar folds.

Eustachian tube:

The Eustachian tube is the conduit through which air is exchanged between the middle ear space and upper aero-digestive tract. It is angled at about 45 degrees from the middle ear to the nasopharyngeal opening at the torus tubaris. The proximal one-third is formed bony petrous part and the distal two-third by fibrocartilagenous portion. The tensor veli palatini muscle is inserted on this membrane retracting it on muscular contraction to open the tubal lumen during swallowing and yawning.

Mastoid air cell system:

Similar to the mastoid, all pneumatized regions of the temporal bone consist of a collection of mucosa-lined bony compartments that are ventilated through the aditus ad antrum or via other cell tracts that open into the middle ear space. This network of bony air cells primarily arises from the diploë of squamous and petrous portions of the temporal bone. The mastoid is the largest pneumatized region, located lateral to the

labyrinth and communicating directly with the attic via its medial compartment, the antrum. The vestibular portion of the inner ear forms the medial wall of the antrum and Körner's septum, the embryologic junction between petrosal and squamosal portions of the temporal bone, forms the lateral limit. Pneumatization may be organized into regions and tracts. The primary regions of pneumatization are as follows:

• The mastoid (the antrum, central mastoid tract, tegmental, sinodural, sinal, facial, and tip cells)

• Perilabyrinthine (supralabyrinthine and infralabyrinthine areas)

• Petrous apex (peritubal area and apical area)

• Accessory region (zygomatic, squamous, occipital, styloid)

3.2.3 The inner ear:

The inner ear is composed of bony and membranous labyrinth and an internal acoustic meatus. Bony labyrinth composed of vestibule, cochlea and semicircular canals.

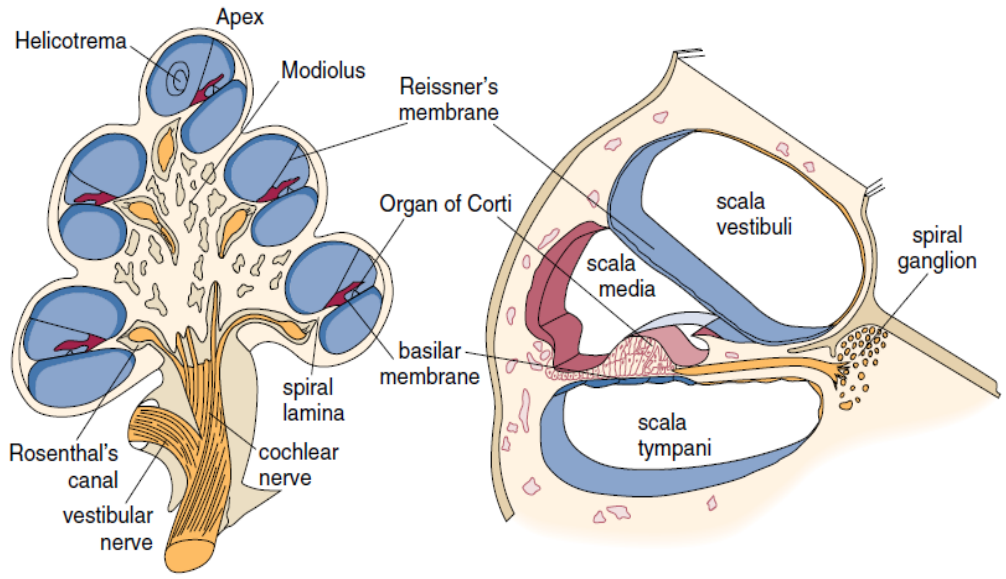


Fig.4.1

Cross sectional structure of the cochlea.

a. Cochlea:

The bony cochlea is a coiled tube making 2.5-2.75 turns round a central pyramid of bone called modiolus. The base of modiolus is directed towards internal acoustic meatus and transmits vessels and nerves to the cochlea. Around the modiolus and winding spirally like a thread of screws, is a thin plate of bone called osseous spiral lamina.it divides the bony cochlea incompletely and gives attachment to the basilar membrane. The bony bulge of the medial wall of middle ear, promontory is due to the basal coil of the cochlea. The body of cochlea contains three compartments; Scala vestibule, Scala tympani and Scala media or the membranous cochlea.

The Scala vestibule and tympani are filled with perilymph and communicate with each other at the apex of cochlea through an opening called helicotrama. Scala vestibule is closed by footplate of stapes which separates from the air filled middle ear. The Scala tympani is closed by secondary tympanic membrane and connected with subarachnoid space through an aqueduct of cochlea.

b. Membranous labyrinth:

The membranous labyrinth of the cochlea follows the shape of the osseous cochlea and forms a third cochlear chamber—the Scala media. The sensory organ of hearing resides within the membranous labyrinth. The membranous labyrinth is bordered superiorly by Reissner's membrane, inferiorly by the basilar membrane, and laterally by a portion of the outer cochlear wall to which it is anchored by the spiral ligament. Within the membranous labyrinth, along the lateral wall, is the stria vascularis, highly vascular tissue that is responsible for the metabolic environment of the Scala media. The intricate structures that comprise the organ of Corti are situated on the basilar membrane. The

organ of Corti runs longitudinally along the length of the basilar membrane and consists of many types of supporting epithelial cells and functional unit structure. Medially seated atop the osseous spiral lamina is the spiral limbus, a thickened band of periosteum that serves as the point of medial attachment for Reissner's membrane and gives rise to the tectorial membrane. The tectorial membrane lies over the inner and outer hair cells. It is a compliant gelatinous structure composed primarily of collagen II fibers. Lateral to the spiral limbus is the inner spiral sulcus, which is lined by the border cells of Held. Single row of inner hair cells is present and the cell bodies are surrounded by supporting cells called phalangeal cells, which surrounded by outer cells with supporting Deiters, Hansen's and Claudius cells laterally. The two fluid systems within the cochlea create an environment crucial to the mechanical displacement of the basilar membrane traveling wave and to the cellular depolarization with subsequent synaptic activity. Between the osseous and membranous labyrinths is filled with perilymphatic fluid or perilymph, which has a high concentration of sodium and low concentration of potassium, similar to what is found in cerebrospinal fluid and blood serum.

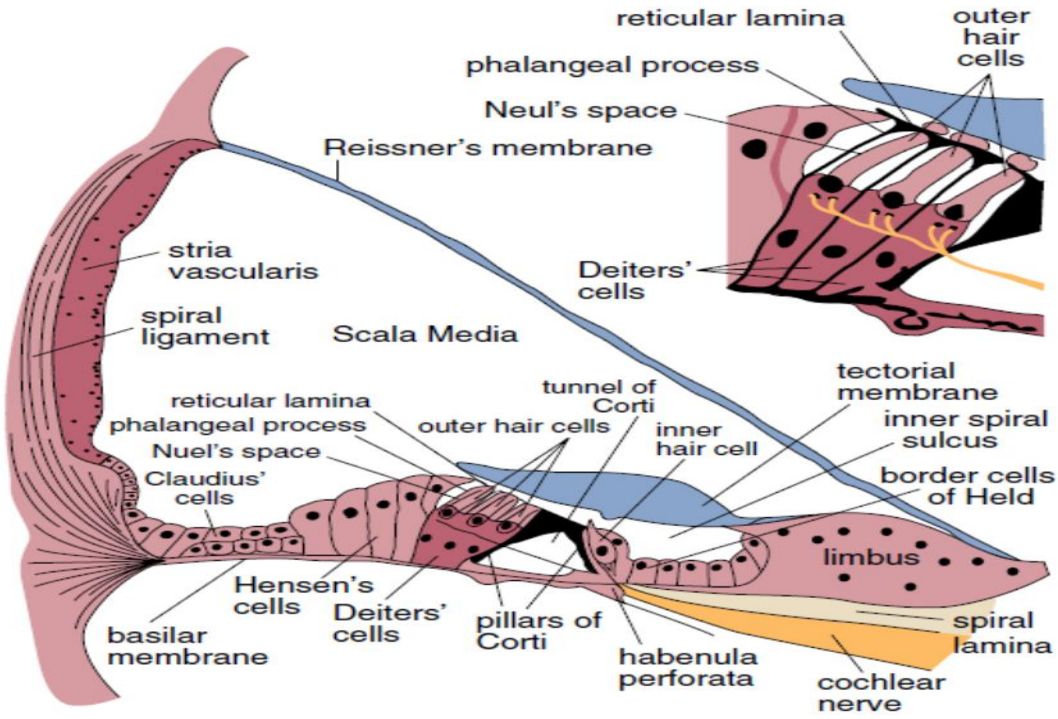


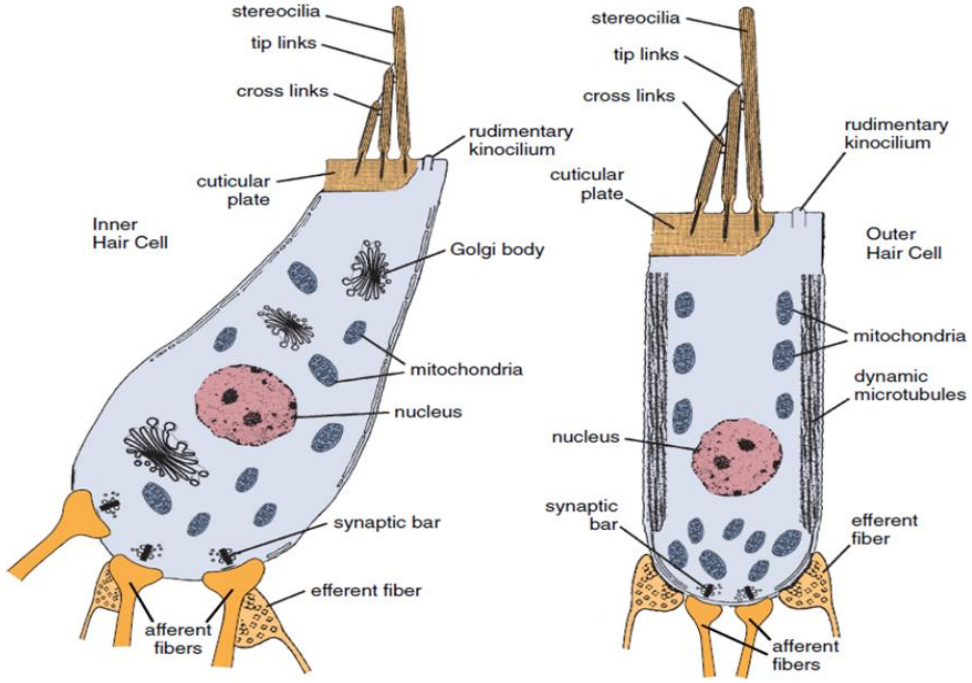
Fig.4.2, Schematic microscopic structure of Scala media.

Within the membranous labyrinth is endolymphatic fluid or endolymph with a high concentration of potassium and low concentration of sodium, such as typically found in an intracellular region. The ionic concentrations of endolymph are maintained by the cells within the stria vascularis. The endolymphatic sac communicates with the membranous labyrinth via the endolymphatic duct and vestibular aqueduct.

Hair cells:

The inner and outer hair cells function as receptor cells that transduce mechanical movement into an electrochemical signal to stimulate the auditory nerve. There are approximately 3500 inner hair cells arranged in one row on the modiolar side of the tunnel of Corti and 12,000 outer hair cells in three rows on the strial side.

Fig.3.3, presents structure of inner and outer hair cells.



The inner hair cells are flask-shaped wide at the bottom and narrower at the top and contain high concentrations of organelles that are involved in metabolic activity, particularly Golgi bodies and mitochondria.

Outer hair cells are cylindrical in shape and contain microfilaments and microtubules along the length of the cell that give rise to motile activity. The motile properties have been shown empirically to result in highly tuned, frequency-specific contractile activity even when stimulated in isolation from the basilar membrane.

The central auditory system

Composed of eighth cranial nerve and auditory projection in brain. The auditory nerve has approximately 30,000 fibers in humans. 90% to 95% of neurons (type I, radial fibers) innervate inner hair cells, whereas 5% to 10% (type II, outer spiral fibers)

innervate to the outer hair cells. These radial fibers have bipolar cell bodies in the spiral ganglion. Outer spiral fibers are monopolar and unmyelinated. The nerve fibers have been classified into three categories on the basis of rate of spontaneous discharge as high (18 to 120 spikes per second), medium (0.5 to 18 spikes per second), and low (0 to 0.5 spikes per second). Fibers with high spontaneous rates have thick dendrites that tend to terminate on the side of inner hair cells facing outer hair cells. Fibers with low and medium spontaneous rates have thin dendrites that terminate on the side of the inner hair cell facing the modiolus. Fibers with a characteristic frequency less than 1 kHz are roughly V shaped here as fibers with a higher characteristic frequency have an obvious tip at the characteristic frequency and a tail that extends to the low frequencies.

Neural pathway:

Eighth nerve end up in the dorsal and ventral cochlear nuclei, axons of which both crossed and uncrossed fibers reaches the superior olivary nucleus, lateral lemniscus, inferior colliculus, medial geniculate body and finally ends up in temporal lobe auditory cortex(Broadman's area 42).

3.3 Physiology auditory system

3.3.1 Physics

Sound and Its Measurement:

Sound is produced by a disturbance in particle density (whereby a particle is made up of many molecules of the sound-propagating medium) triggered by a sound-producing body or sound source. The velocity of sound propagation in dry air is about 340 m/sec at room temperature, whereas in water sound travels at the rate of 1500 m/sec.

The *frequency* of a simple harmonic motion is the number of cycles per second and is measured in *Hertz* (Hz). The *period* of a cycle is the inverse of its frequency (1/*f*), and represents the duration of a single cycle. The *amplitude* is the maximum amount of displacement from the null point in one direction. The sound produced by simple harmonic motion is called a *pure tone*. Any vibration that does not follow simple harmonic motion is said to be complex. If the complex vibration has no repetitive pattern, it results in *noise*. One way to quantify sound is by its intensity, which is cumbersome to measure directly. Sound *pressure,* which is related to the square root of intensity, is relatively easy to measure, however, and is the most common way of quantifying sound.

A convenient way of expressing Sound intensity is by taking the logarithmic ratio of two sound intensities and multiplying by 10. This is called the *decibel scale*. The formula for determining decibels for sound intensity is:

dB = 10 log10 J/Jr,

Where *J* is the intensity of the sound of interest, and *Jr* is the intensity of reference.

The formula for determining decibels for sound pressure is

dB = 10 log10 P2/Pr2 = 10 log10 (P/Pr)2 = 20 log10 P/Pr

Where **P**-is the sound pressure of interest

Pr-is the reference sound pressure.

Loudness measured as sound pressure level

The most commonly used reference sound pressure is 20 μPa,

Which is referred to as *sound pressure level* (SPL). Another reference sound pressure that is occasionally used is *hearing level* (HL).

Impedance:

In the study of acoustics, impedance is defined as the ratio of the acoustic pressure over the volume velocity generated by the acoustic pressure. There are three components to acoustic impedance: stiffness, resistance (damping) and mass.

ABSOLUTE THRESHOLDS:

The absolute threshold for a sound is the minimum detectable level of the sound in the absence of any other background sound or noise. Threshold are measured in two ways MAP (minimum audible pressure) and MAF (minimum audible field). However, the general form of the results is similar that of "normal listeners" may have thresholds up to 20 dB above or below the average. Thresholds tend to increase with age, particularly at high frequencies (4 kHz and above). The range of frequencies where sensitivity is greatest for human ear is 500-5000 Hz, also the range most important for understanding speech.

THE PERCEPTION OF PITCH:

Pitch is a subjective sensation produced by frequency of sound; the higher the frequency, the higher the pitch.

Hearing theory:

1. Place Coding Theories

- Resonance theory (Helmholtz, 1885)
- Traveling wave theory (Georg von Bekesy 1928-1958- Nobel Prize 1961)

- Many other theories including standing wave theory, pressure pattern theory, frequency analytic theory, etc. See Zemlin for details.

2. Temporal Coding Theories (frequency theory)

- Telephone theory (Rutherford, 1886)
- Volley theory (Wever, 1949).

For pure tones, pitch perception and discrimination are determined primarily by temporal information for frequencies below 4-5 kHz, and by place information for frequencies above this. The important frequencies for the perception of music and speech lie in the frequency range where temporal information is available.

Loudness:

It is the subjective sensation produced by intensity of sound; more the intensity of sound, more the loudness.

Recruitment:

Cochlear hearing loss often results in an abnormality of loudness perception known as loudness recruitment. The absolute threshold is elevated, but for high sound levels (around 90-100 dB SPL) the sound appears as loud in the impaired ear as it would in a normal ear.

Decibel:

It is $1/10^{th}$ of a bel and named after Alexander Graham Bell.it represents logarithmic ratio of two sounds. In audiology sound is measured as Sound pressure level (SPL) in decibels (dB).

Noise

Defined as aperiodic complex sounds or a nonharmonic sound.

White noise: Noise that contains all frequencies in audible spectrum. Broad band noise used for masking.

Narrow band noise: It is a white noise with certain frequencies above and below the given noise, filtered out.

Speech noise: it is noise having frequencies in the speech range (300-3000 Hz).

Audiometric zero:

According to ISO, the audiometric zero is the mean value of minimal audible intensity in a group of normal hearing healthy young adults.

4.3.2 Physiology of External Ear;

The external ear funnels sound from the external environment into the ear. The concha has a resonant frequency of around 5300 Hz, and the external auditory canal has a resonant frequency of around 3000 Hz. The external ear plays an important role in sound localization. Sound localization is achieved by two major mechanisms: interaural time difference and interaural amplitude difference.

4.3.3 Middle ear mechanics;

As a sound stimulus enters the external auditory canal, it causes the tympanic membrane to vibrate. The malleus, which is coupled to the tympanic membrane, vibrates in response to the motion of the tympanic membrane. This causes the entire ossicular chain

to vibrate, resulting in sound transmission to the inner ear via the stapes footplate is referred to as *ossicular coupling.* The pathway of sound transmission to the inner ear in the absence of the ossicular system is referred to as acoustic coupling. It has been shown that the difference between ossicular coupling and acoustic coupling is about 60 dB, which is the maximal amount of hearing loss expected in patients with ossicular discontinuity Middle ear can be explained in two way impedance matching and lever ratio. The overall middle ear gain is about 20dB.

4.3.4 Inner ear physiology;

The inner ear is enclosed in a bony cavity called the otic capsule. It has two mobile windows: the oval and the round windows**.** The two important functions of the inner ear are hearing and balance. The portion of the inner ear that deals with hearing is the cochlea.

The unique electrolyte composition of the Scala media sets up a large electrochemical gradient, called the *endocochlear potential*, which is +60 to +100 mV relative to the perilymph. The maintenance of such a large electrochemical gradient is performed by the *stria vascularis*, which resides on the outer wall (away from the modiolus) of the Scala media. The stria vascularis contains multiple active ion channels, and maintains the chemical composition of the endolymph and its positive electrical potential.

As sound energy travels through the external and middle ears, it causes the stapes footplate to vibrate. The vibration of the stapes footplate results in a compressional wave in the inner ear fluid, which travel across the Scala vestibule, around the

helicotrema, and out across the Scala tympani toward the round window. An inward motion of the Stapes causes an outward motion of the round window. As this compression wave travels across the Scala vestibule, however, the pressure in the Scala vestibuli is higher than the pressure in the Scala tympani. This sets up a pressure gradient, which causes the cochlear partition to vibrate. Von Bekesy12 first described the vibration of the cochlear partition in cadaveric human cochleas. He showed that as the cochlear partition is deflected by the compressional wave created by the stapes footplate vibration, it sets up a traveling wave on the basilar membrane, which travels from the base of the cochlea to its apex. The basilar membrane is *tonotopically tuned* to different frequencies along its length.

As the cochlear partition is deflected in response to the compressional wave initiated by the stapes, it causes a shearing force between the stereo cilia of the hair cells and the tectorial membrane. This shearing force causes a deflection of the hair cell stereo cilia. The hair cell stereo cilia are arranged in rows, and the rows are arranged in an orderly fashion by height. The tip of each stereo cilia is connected from one row to another by an elastic filament called the *tip link.* It is thought that as the stereo cilia is deflected toward the direction of the tallest row, it causes the tip links to stretch. The stretch of the tip links causes the opening of stretch-sensitive cationic channels located on the stereo cilia.

Because there is a large electrochemical gradient across the apical surface of the hair cells, the opening of these stretch-sensitive cationic channels on the stereo cilia causes a large influx of cationic current, which leads to hair cell depolarization. Stereo cilia deflection in the depolarization direction produces a greater response than deflection in

the hyperpolarization direction. The deflection of the hair cell stereo cilia and the resulting hair cell depolarization or hyperpolarization represents an important step in the signal transduction process of the hair cell.

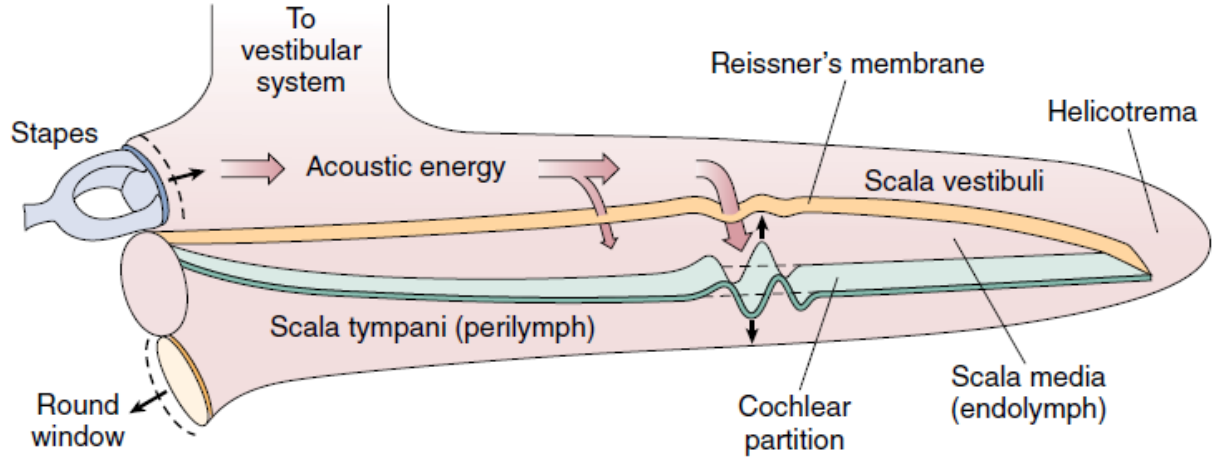


Fig 4.4, schematic presentation of sound conduction in inner ear.

Since potassium is the major cation in the endolymph, it is believed that potassium current plays an important role in triggering the signal transduction process in hair cells. When inner hair cells are depolarized, voltage-gated calcium channels open.3. These voltage gated calcium channels are clustered in several "hot spots" along the basolateral surface of the inner hair cells, where synaptic contacts with primary afferent auditory nerve fibers are located.3, 4. The calcium current mediated by these voltage-gated ion channels are important for triggering neurotransmitter release across the synapse, which leads to activation of the auditory nerve fibers. The neurotransmitter involved in this process has not been definitively identified, but is believed to be a molecule closely related to glutamate.

In contrast to the inner hair cell, an outer hair cell can also change its length in response to voltage changes; it contracts with depolarization and elongates with hyperpolarization.3, 2. The molecular motor that is associated with rapid changes in outer hair cell length is a voltage dependent, integral membrane protein called prestin.(2, 3and 4) The change in outer hair cell length in response to voltage changes is believed to add energy into the basilar membrane motion through a mechanical feedback scheme. In other words, the outer hair cell acts as a cochlear amplifier, augmenting the signals transmitted into the inner ear by the stapes vibration.

Different regions of the basilar membrane are tonotopically tuned to specific frequencies, because the hair cells reside on top of the basilar membrane is logical to assume that the hair cells from different regions are also tonotopically tuned to specific frequencies.

4.3.5 Auditory Nerve:

When the mechanical vibration of the cochlear partition is transduced into electrochemical signals by the hair cells, the information carried by these signals is propagated by the afferent auditory nerve fibers into the brain for processing across neuronal stations and pathways. The afferent auditory neurons are bipolar in nature, sending peripheral processes to make contact with hair cells, while sending central projections to the auditory brainstem. Their cell bodies, also known as the spiral ganglion cells, are located in Rosenthal's canal. Humans have approximately 30,000 spiral ganglion cells. There are two types of spiral ganglions. Type-1 myelinated accounts 90% and type 2 unmyelinated 10%. Each type 1 ganglion synapses with single

inner hair cells, whereas type2 synapses with multiple outer hair cells. As the sound pressure increases over a certain level, the afferent neurons begin to discharge at a higher rate than the spontaneous rate. This sound pressure level is called the threshold. The relationship between sound pressure level and threshold is a nonlinear one, however, because as the sound pressure increases over a certain level, the discharge rate of the afferent neurons begins to saturate. The difference between the sound pressures at threshold and at saturation is called the dynamic range of the afferent neuron.

The high spontaneous neurons are important for detecting low-level sounds (Because of their low threshold), whereas low spontaneous neurons are important for signaling amplitudes at high level sounds (Because of their large dynamic range).

At a given location along the cochlear partition, the basilar membrane, the hair cells, and the afferent neurons all have the same characteristic frequency. As a sound stimulus enters the cochlea, its frequency components are analyzed by the basilar membrane as a series of filters. This frequency information is preserved through the hair cells and the auditory afferent neurons, and is transmitted to the central nervous system. The threshold sound pressure for auditory neurons can be (−10 dB SPL) which is equivalent to 7 μPa.

Cochlear Nucleus:

The auditory nerve travels along the course of the internal auditory canal to terminate on the second-order neurons of the auditory system located in the cochlear nucleus. The cochlear nucleus is the critical first relay station for all ascending auditory information

originating in the ear, and is located in the Ponto medullary junction of the dorsolateral brainstem in humans. The cochlear nucleus contains many cell types, each having unique somatic and dendritic characteristics. The distribution of cell types subdivides the cochlear nucleus into its major subdivisions: the dorsal cochlear nucleus, the anterior ventral cochlear nucleus, and the posterior ventral cochlear nucleus.

The second-order neurons of the cochlear nucleus are tonotopically organized. The spatial representation of frequency-specific information in the cochlea is preserved in the cochlear nucleus. Iso-frequency lamina (sheets of neurons that have the same characteristic frequency) are distributed from dorsal to ventral across each major cochlear nucleus subdivision, and are seen in higher auditory nuclei.

Inputs from auditory nerve drive multiple cell types in different subdivisions of the cochlear nucleus, with each cell type projecting centrally to different targets in the superior olivary complex, lateral lemniscus nuclei, and inferior colliculus. Because individuals with normal hearing use both ears, sound localization is accomplished by neural processing of intensity and timing cues from each ear in the auditory brainstem. The temporal and spectral features of sound originating in the ear are processed in the cochlear nucleus, and the cochlear nucleus is the origin of parallel pathways. These pathways that project to the auditory brainstem, midbrain, and cortex integrate information from the ear to determine as follows,

(1) The identity of the sound source,

(2) The intensity of the sound source,

(3) The location of the sound source.

The ventral cochlear nucleus contains many different cell types:

(1) Spherical bushy cells found primarily in the Anteroventral cochlear Nucleus (rostral),

(2) Globular bushy and multipolar cells found centrally,

(3) Octopus cells found posteriorly (caudal).

Superior olivary nucleus;

Auditory information from both cochlear nuclei is integrated in the superior olivary complex; this region plays an important role in sound localization by analyzing interaural time and amplitude differences.

Lateral Lemniscus;

The lateral lemniscus is formed by the three fiber tracts from the cochlear nucleus. It acts as collateral between superior olivary and contralateral inferior colliculus.

Inferior colliculus;

Anatomic and physiologic studies show that the inferior colliculus receives auditory inputs from the lateral lemniscus, the cochlear nucleus, and the superior olivary complex, and projections from the somatosensory system, and the visual and vestibular systems. The inferior colliculus processes the information it receives and sends fibers to the medial geniculate body of the thalamus.

Medial Geniculate Body;

The medial geniculate body is the thalamic auditory relay center that receives auditory information from the inferior colliculus. From here it projects to the primary auditory cortex Brodmann's area41, and association cortex 42, which play an important role in sound localization and processing of complex vocal communications, such as human speech.

The primary auditory cortex is located on the superior surface of the temporal lobe (Heschl's gyrus). This is also known as area A1, and corresponds to Brodmann's area 41. The auditory association cortex is also known as area A2, and corresponds to Brodmann's areas 22 and 42. The primary auditory cortex is involved with integrating and processing complex auditory signals, including language comprehension.

The auditory association cortex is located lateral to the primary auditory cortex, and it is part of a language reception area known as Wernicke's area. In addition to above projections are sent to limbic system too.

4.4 Pathology

Noise induced hearing loss:

Definition: The term NIHL refers to the reduction of auditory acuity due to exposure to noise.

Temporary threshold shift (TTS):

TTS refers to reversible NIHL, although time duration is not available may be hours to a day after which a person may regain his hearing. Noise exposure of 78dB would not produce TTS (ISO 1999 indicates 75 dB as risk threshold). But above 80db produces TTS with very short life.

Permanent threshold shift (PTS);

The NIHL may be permanent without regaining hearing even after a day refers to PTS. PTS may develop due to repeated TTS or following single impulse exposure (acoustic trauma). The precise relationship between TTS and PTS stages not known. Two types of PTS are classified one the acoustic trauma (due to intense noise exposure) and the other NIHL. But there no studies duration repeated TTS that causes PTS.

Noise and its measurement:

Noise literally means an undesirable sound (unpleasant sound). Characteristically may be classified into continuous, fluctuating, intermittent, or impulsive. Continues are those relatively constant, whereas fluctuant increase and decreases over time and intermittent varies in on off pattern. Impulsive accounts for short lasting followed by reverberations and echoes, noise caused by explosives, metal on metal collision etc. noise can be measured by personal dosimeters as sound pressure level in dB units. Current permissible level of continues noise exposure is less than or equal to 85dB lasting for 8hrs.

Pathology:

Pathological changes due to NIHL (vibration associated) are still under debate, though majority of studies consider cochlear function. The primary site of damage due to NIHL is the sensomechano receptors, an inner and an outer hair cells. Outer HC are the first to suffer due high intensity noise followed with inner cells. The high resolution microscopic study reveals hair cells and nerve degeneration at frequency range of 4 KHZ and progress towards high frequency and mid frequency of basal turn. Although low frequency hair cells are intact patient may not able perceive sound due to PTS. The mechanism under this logic is still unknown. Two mechanisms of cochlear damage were evident so far.

I. Metabolic mechanism

NIHL is a multifactorial and complex situation. Noise leads to the excessive glutamate release due to over stimulation receptors, which evidenced by administration of receptor antagonist reduces the incidence of TTS (3, 4). There are animal studies specifically stimulation with moderate intensity sound increases cochlear blood flow and vice versa for high intensity sound, which contributes to hypoxia of cochlea. Some other mechanism focused in experimental studies are OHC plasma membrane fluidity, oxidative stress and the role of glucocorticoids.

II. Structural mechanism:

Changes in the micromechanical structures may be a contributing factors for NIHL. Factors are depolymerization of actin filaments in steriocilia, swelling of stria vascularis, afferent nerve endings and other supporting cells are noted microscopically.

III. Apoptosis and necrosis:

Animal based studies evidenced the OHC condensation after few minutes of noise exposure and OHC necrosis after 30 min of exposure were noted. A present study goes on cysteine dependent aspartate specific protein in OHC apoptosis. More recent studies are evident that hair bundles are capable of rebuilding their unit within 48 hrs. It is also evident that in severe damage it overwhelms it regenerating capacity by direct mixture of endolymph and perilymph by micro injury at specified frequency space.

Predisposing factors: NIHL is influenced by many factors

a) Genetic factor Ahl gene

b) Age factor

c) Diabetes anemia and cardiovascular disease

d) Smoking

e) Ototoxicity and recreational drugs

f) Environmental factors that causes stress in working place

(Vibration –very few studies).

4.5 Diagnosis of NIHL:

Introduction:

There is a vast biological variability and individual susceptibility to noise, as well as insidious nature of progress of NIHL. It means that by the time the individual presents with complaint exposure to noise would have ceased and reached age factor for hearing impairment.so it very difficult to conclude for an accurate diagnosis. For practical study purpose it is better to start with the assumption of diagnosis in specific group composed of three groups an age related group, a noise induced component and the idiopathic degenerative component. The observer task is to calculate the relative contributions from the three groups.

Acoustic shock is of different entity develops due acute intense noise exposure which is not included in this study. Only the clinically healthy subjects are considered for my study purpose.

History:

a) Age
b) Family history of early hearing loss
c) Ear discharge, ear pain, sense of block, HOH, loudness intolerance
d) Noise exposure, duration, exposure to intense noise, different or similar noise exposure
e) Presence TTS, tinnitus,

f) Diabetes, hypertension, cardiovascular disease, Reynaud's phenomena, head injury, meningitis

g) Medication particularly aminoglycosides, thiazide diuretics

h) Smoking, alcohol and abusing substance habits

Symptoms:

1. Tinnitus
2. Loudness intolerance
3. Temporary threshold shift

Acoustic shock:

a) Otalgia

b) Tinnitus

c) Hyperacusis

d) Dizziness

e) Headache

f) Sleep disturbance

g) Poor concentration.

Examination:

Ear: pinna, EAC, presence of wax, tympanic membrane (otoscopic) and mastoid process (three finger test).

Differential diagnosis

a) Presbyacusis which includes midrange frequency loss in relation to the progressive age that can be ruled out by comparing to the standard age related hearing loss chart

b) Conductive hearing loss: if present always caused by some other degenerative causes to be sought out.

c) Idiopathic Sudden sensoneural hearing loss: ISSHL is characterized by acute onset with 30 dB sensorineural loss in ≥3 contiguous frequencies in < 3 days duration.

d) Ototoxicity: cochlear damage caused by chemical injury as a side effect of pharmacotherapy. This can be ruled out by treatment history after which hearing loss developed.

Investigations:

a) Random blood sugar, blood pressure and hemoglobin

b) Tuning fork test: Rinne's, Weber's, ABC test.

c) Pure tone audiogram: Air conduction and bone conduction for octave

Frequencies (250hz, 500hz, 1khz, 2khz,4khz,8khz)

PTA though a subjective test, is the gold standard to diagnose NIHL. The classical 4-6 k notching can be identified in PTA for NIHL with symmetrical graph for both sides and without involvement of mid low frequencies preserving speech magnitude.

d) Oto acoustic emission test OAE

This is ideal test for assessing normal functioning of cochlea, which depends on functions of OHC.OHC are the one that is involved in the primary stage of NIHL. Enough studies have evidenced that humans exposed to noise have reduction in OAE, more sensitive than PTA. Additionally OAE detects more accurate frequency

magnitude and symmetrical involvement than PTA in the early stages, which is not so in PTA.

Diagnosis of NIHL is simple in an individual with a prolonged history of exposure to noise and no evidence of any other otological problem with evidence of PTA showing preservation of speech frequency with significant high tone hearing loss and classical notch.

4.6 Management:

The first and foremost thing in the management of NIHL is prevention by protection. Because by the time we diagnose the individual would have reached his older age and further there is no treatment for sensorineural hearing loss (NIHL).Though there are studies going on for regeneration of hair cell with improved hearing function evidenced in animals.so insisting on personal protection with ear plugs or muffs, avoidance of further exposure to noise is the major solution so far to date.

The employee must conduct a noise survey and implement an occupational noise and vibration management program.ie

- Identifying the risk hazard and assessment,
- Hearing conservation policy statement,
- Surveys for noise level exposure and control measures by engineering
- Conduct a program of employee education and training.
- Provide appropriate hearing protection, the use of which is at discretion of the employee until the peak level is reached when it becomes compulsory.

- Regular hearing (audiometric) test should be offered to the employee.
- Reduction of noise level at source.
- Assessment of effectiveness of the above program and record maintenance.

Daily vibration exposure:

- Action(EAV); 2.5m/s^2 and
- Limit (ELV); 5m/s^2 for hand arm vibration.
- EAV;0.5 m/s^2 and
- ELV; 1.15m/s^2 for whole body vibration.

Personal hearing protection

- Ear plugs 10-15 dB attenuation.
- Ear muffs at least 15dB attenuation.
- Electronic sound attenuation effectively clears out background noise, but expensive.
- Unfortunately variability in susceptibility to noise damage, adherence to all standards does not guarantee protection.
- Noise level of 80 or less will not produce hearing impairment even after years of exposure (**Robinson *et al* 1994).** Above 90dB this becomes worse and progressive.

Nonspecific management:

There is no way to replace the hearing loss except supportive with Hearing aids, lip reading, psychological counselling and acceptance of their problem in public.

Specific management;

- Digital or analogue Hearing aid.
- Tinnitus retraining therapy.
- Hair cell regeneration and repair: generally hair cell loss is irreversible, but recent in vitro animal studies has an evidence for regeneration and functional improvement by injecting an adenovirus carrying Math 1 gene.

Table ;3.1 shows permissible and measured value as per Indian automobile Amendment Rules, 2000, vide G.S.R. 742 (E), dated 25th September, 2000.

Vehicle type	**Observed value dB(A)**	**Allowed dB(A) from year 2003 (implemented)**	**Vibrations measured(0.1-1m/s^2) In our study**
Auto-Rickshaws	81-96	77-80	
Trucks	83-90	80-90	
Buses	80-92	80	0.2-9.0m/s^2 y axis
Cars	72-80	75	

Table; 3.2 frequency loss (NIPTS) for corresponding sound pressure level. (57)

Frequency kHz	0.5	1	2	3	4	6
***L*0, dB**	93	89	80	77	75	77

Hearing threshold can be derived from $H = A + N - [(A \times N) / 120]$.

Where A is age related loss, N is NIPTS from above table in dB (57).

To obtain the value of whole body vibration and noise effects, instruments called dosimeter and accelerometer sensor is used.one of the randomly selected bus was measured using instrument from nearby engineering college student help. Accelerometer sensor is a device to measure the vibrations. This device is placed on the driver seat. Later, the bus driver would sit on the equipment along the way to the destination. Then, all data will be entered into a program at Quest Software to make graphs. Overall, the vibrations are recorded maximum for y-axis 9.5888 m / s ². Data's were compared with data's from ISO 2631. According to ISO 2631, for the time worked 4hours, RMS value is 4 m / s ². Thus, the value obtained exceed the value of ISO 2631. Noise measured in the range of 70dB -95dB for about an hour exceed 92dB.

Methodology

In this cross-sectional study we study the effect of vibration with noise indirectly on hearing in drivers, who are in regular contact with engine control. Conductors are taken as control group, who are exposed to noise in similar to drivers but not to vibrations as drivers.

Study mainly based on pure tone audiometry data from otologically normal driver and conductors from a selected transport corporation.

Data for this study was collected from the department of ENT head and neck surgery, from those referred from state transport corporation for general medical health checkup from October 2014 to September 2015 and by requesting their friends from other places but from only state transport corporation employees so that uniform exposure all subjects to the noise environment can be maintained.

103 patients were screened and selected for this study. All patients fulfilled our study criteria.

Selection criteria: Subjects those who are clinically healthy are selected

Inclusion criteria:

a) Subjects with minimum of 5yrs service as driver or conductor
b) Subjects only with informed consent and only from State transport corporation. (TNSTC&SECTC)

c) Subjects who drives more than 500 kms per day .e.g. Those who performs double and triple attendance.

Exclusion criteria:

a. Patients with any ear pathology.
b. Patients those who have undergone ear surgery.
c. Congenital ear deformity and family positive history of HOH by birth.
d. Patients with systemic illness that affects hearing (diabetes, hypertension, cardio vascular disease, neurological disorders of more than 6 months).

A thorough history regarding age, sex, complaints, past illness, family history, smoking, alcohol and exposure to intense sound were enquired before the subject is taken for study. Since most of our subjects are habituates of alcohol (weekly, monthly and rarely), smoking and pawn are unable to include in exclusion criteria difficult to get pure subjects for the study.

Complete ear examination were done and recorded in proforma. Investigations like Pure tone audiogram, hemoglobin, blood urea, serum creatinine, random blood sugar and tuning fork to rule out conductive deafness any discordant hearing in either ear were done.

Tuning fork test (Rinnes, webers and absolute bone conduction) were performed to rule out conductive hearing loss for exclusion from study. All history and findings were recorded in proforma (attached to annexure).

Pure tone audiogram;

Digital computer based two channel clinical Audio lab manufactured by LABAT Italy with proper calibration according with standards of ISO 389-7 (1996) were used in proper acoustic room less than 20dB using head phone TDH49. The maximum background sound pressure level in the testing room was followed as per table 4.2, by using typical supra-aural earphones such as the Telephonic TDH39/49 with MX 41/AR cushions.

Conventional method (Hughson-Westlake) of audiogram recording was done. Test begins with 1000 Hz followed with 2 kHz, 4 kHz, and 8 kHz again followed by 500Hz and 250Hz starting from 50 decibels reducing by 10 and increasing by 5 dB intervals. Subjects with more than 40db loss difference between right and left were considered as exclusion criteria and not included in the study.

To avoid the temporary threshold shift PTA was done with the minimum gap of 12 hrs. From sign off duty. Testing condition and environment were strictly followed as per Norms

So there are no masking criteria for this study, though tested in some subjects for exclusion criteria. Web based master calculator

http://www.occupationalhearingloss.com/master_calculator.html are used to eliminate age related hearing loss, being the most confounding factor for the study was calculated by Robinson and Sutton's formula (68)and NIOSH 1998 for the final hearing impairment in percentage by noise and vibration effect. Hearing loss was also calculated separately for high and low (speech) frequency by conventional method;

Hearing loss for right or left ear % = (A-25) ×1.5

Where A=a+b+c / 3, a, b, c, are loss for frequency (LFL-500Hz, 1 kHz &2 kHz) &

(For HFL-4 kHz, 6 KHz &8 kHz) respectively.(adapted from ISO: R 389-1970)

Total percentage of loss by (5x +y)/6. Where x=worst ear loss and y=better ear, since attenuation of better ear is 5 times that of worst ear.

Hearing loss by age factor (Robinson &Sutton's) =a $(N1-18)^2$+b,

Where a and b are frequency dependent dispersion parameters as in table Annexure.

All data's were analyzed by using chi-square test, t-test and paired t-test for comparing drivers and conductors in respect to their age, service (duration of exposure),right and left ear for LFL,HFL and overall hearing loss by master calculator using NIOSH1998 and Robison and Sutton formula (approved for study purpose by OSHA).

The results are interpreted and discussed in comparison with other related journals.

Groups are classified as per WHO classification as follows:

. Table 4.1 WHO classification for degree of hearing handicap

dB HL	*range Hearing loss category*
0-25	Normal hearing
26-40	Mild hearing loss
41-55	Moderate hearing loss
56-70	Moderately severe hearing loss
71-91	Severe hearing loss
>91	Profound hearing loss

Table: 4.2, Maximum permissible ambient noise levels PTA with 5 dB uncertainty over the range 500-8,000 Hz, using typical supra-aural earphones such as the Telephonic (adapted from ISO 8253-1).

Octave band Centre frequency (Hz)	**Maximum permissible background sound pressure levels LS, max (dB re 20 µPa)**	
	Test tone frequency range (Hz)	
	Air conduction audiometry	**Bone conduction audiometry**
	500- 8,000	**500 - 8,000**
125	55	33
250	46	24
500	31	31
1000	33	20
2000	40	19
4000	47	15
8000	46	22

RESULTS

Table.5.1, Chi-Square Tests

	Occupation						Statistical Inference
	Conductor		Driver		Total		
	(n=50)	(100%)	(n=50)	(100%)	(n=100)	(100%)	
Age							
Below 40yrs	11	22.0%	20	40.0%	31	31.0%	X^2=12.403 Df=2 .002<0.05 Significant
41 to 50yrs	16	32.0%	23	46.0%	39	39.0%	
51yrs & above	23	46.0%	7	14.0%	30	30.0%	
Service							
Below 10yrs	9	18.0%	14	28.0%	23	23.0%	X^2=8.735 Df=2 .013<0.05 Significant
11 to 20yrs	17	34.0%	26	52.0%	43	43.0%	
21yrs & above	24	48.0%	10	20.0%	34	34.0%	

Fig 5.1, Age vs Occupation

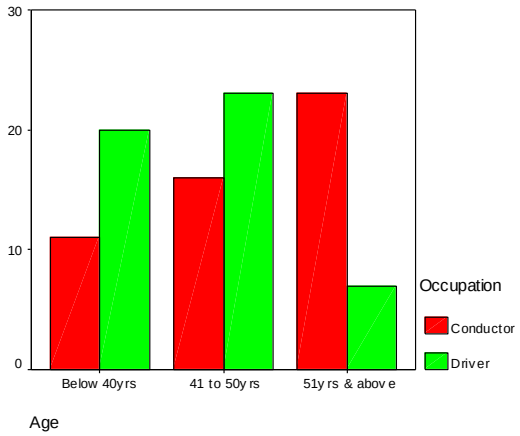
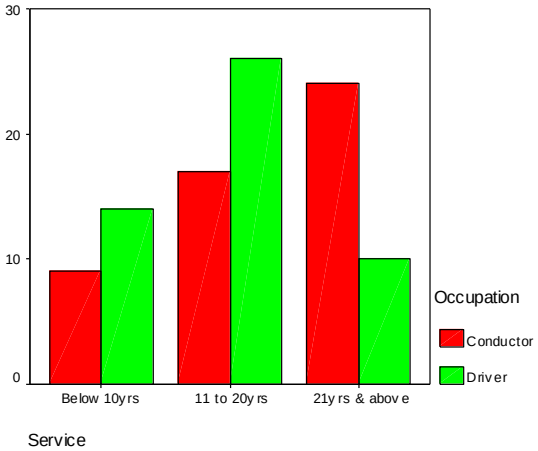


Fig 5.2, Service vs Occupation



The p-value is significant for the age and service distribution in our study.

Table 5.2, T-Test results.

	Mean	S.D	Statistical inference
Age			
Conductor (n=50)	48.38	6.954	T=3.670 Df=98 .000<0.05 Significant
Driver (n=50)	43.40	6.612	
Low Frequency (Left)			
Conductor (n=50)	8.950	11.5185	T=-.089 Df=98 .929>0.05 Not Significant
Driver (n=50)	9.150	10.9452	
Low Frequency (Right)			
Conductor (n=50)	8.950	12.1962	T=-3.536 Df=98 .001<0.05 Significant
Driver (n=50)	21.100	21.0148	
Low Frequency (Total)			
Conductor (n=50)	6.520	8.1924	T=-1.920 Df=98 .050=0.05 Significant
Driver (n=50)	10.409	11.7438	
High Frequency (Left)			
Conductor (n=50)	20.450	16.4727	T=-.172 Df=98 .864>0.05 Not Significant
Driver (n=50)	21.100	21.0148	
High Frequency (Right)			
Conductor (n=50)	18.800	16.5619	T=-.645 Df=98 .520>0.05 Not Significant
Driver (n=50)	21.450	23.8463	
High Frequency (Total)			
Conductor (n=50)	15.814	13.1269	T=-.887 Df=98 .377>0.05 Not Significant
Driver (n=50)	18.892	20.7186	
MCL			
Conductor (n=50)	11.5568	9.53017	T=-.892 Df=98 .375>0.05 Not Significant
Driver (n=50)	13.7430	14.48163	
Service			
Conductor (n=50)	20.00	8.266	T=3.282 Df=98 .001<0.05 Significant
Driver (n=50)	15.32	5.776	

The above Table 5.2 shows non significance for MCL hearing loss because there is no major difference but minor difference of about 3 dB loss more in drivers compared to conductors are alike as in many studies. In the same way difference in the high frequency loss too around 3dB, but there is a major difference between the drivers and

conductors for low frequency loss with significant p-0.050.this shows that drivers are earlier to get affected by noise in low frequency following HFL.

Table 5.3 Paired sample T-Test for conductors (n=50)

	Mean	S.D	Correlation	Sig.	Mean	Std. Deviation	t	df	Statistical inference
Pair 1									
Low Frequency (Left) (n=50)	8.950	11.5185	.415	.003	-11.500	15.6980	-5.180	49	.000<0.05 Significant
High Frequency (Left) (n=50)	20.450	16.4727							
Pair 2									
Low Frequency (Right) (n=50)	8.950	12.1962	.416	.003	-9.850	15.9672	-4.362	49	.000<0.05 Significant
High Frequency (Right) (n=50)	18.800	16.5619							
Pair 3									
Low Frequency (Total) (n=50)	6.520	8.1924	.409	.003	-9.294	12.3030	-5.342	49	.000<0.05 Significant
High Frequency (Total) (n=50)	15.814	13.1269							

Table 5.3 shows significant p-value for comparison between high and low frequencies. This shows that conductors are affected in noise pollution with NPTS.

Table 5.4 Paired sample T-Test for Drivers (n=50)

	Mean	S.D	Correlation	Sig.	Mean	S.D	t	df	Statistical inference
Pair 1									
Low Frequency (Left) (n=50)	9.150	10.9452	.615	.000	-11.950	16.6927	-5.062	49	.000<0.05 Significant
High Frequency (Left) (n=50)	21.100	21.0148							
Pair 2									
Low Frequency (Right) (n=50)	21.100	21.0148	.899	.000	-.350	10.4370	-.237	49	.814>0.05 Not Significant
High Frequency (Right) (n=50)	21.450	23.8463							
Pair 3									
Low Frequency (Total) (n=50)	10.409	11.7438	.795	.000	-8.483	13.4232	-4.469	49	.000<0.05 Significant
High Frequency (Total) (n=50)	18.892	20.7186							

Table 5.4 shows the significant p-value for the comparison between LFL and HFL in drivers, which indicates drivers are affected with occupational noise induced hearing loss (NPTS).

Table 5.5 Descriptive statistics of the whole study

Items	Both (n=100)				Conductor (n=50)				Driver (n=50)			
	Min.	***Max.***	***S.D***	***Mean***	***Min.***	***Max.***	***S.D***	***Mean***	***Min.***	***Max.***	***S.D***	***Mean***
Age	32	57	7.200	45.89	35	57	6.954	48.38	32	56	6.612	43.40
Low Frequency												
Left	-2.5	52.5	11.1791	9.050	-2.5	52.5	11.5185	8.950	.0	45.0	10.9452	9.150
Right	.0	90.0	18.1516	15.025	.0	62.5	12.1962	8.950	.0	90.0	21.0148	21.100
Total	-2.1	45.8	10.2616	8.464	-2.1	44.2	8.1924	6.520	.0	45.8	11.7438	10.409
High Frequency												
Left	.0	90.0	18.7881	20.775	.0	70.0	16.4727	20.450	.0	90.0	21.0148	21.100
Right	.0	107.5	20.4692	20.125	.0	70.0	16.5619	18.800	.0	107.5	23.8463	21.450
Total	.0	92.9	17.3246	17.353	.0	50.8	13.1269	15.814	.0	92.9	20.7186	18.892
Others												
MCL	.00	45.00	12.24582	12.6499	.00	42.50	9.53017	11.5568	.00	45.00	14.48163	13.7430
Service	6	31	7.474	17.66	6	31	8.266	20.00	8	28	5.776	15.32

The above table shows the data analysis of whole study in a brief. Here we can see the maximum loss for drivers is 107dB and for conductors it is only

70dB. ISO 1999 90 and above is considered as 100 % loss which criteria is met by drivers for 4 kHz frequency (4K dip in audiogram).

Table 5. 6. Two tailed p-value for service with hearing loss

Duration of service driver and conductor	LFL	HFL
Less than 10 years	0.009 sig	0.419 Not sig
10-20 years	0.067 Not sig	0.082 Not sig
20 years. and above	0.039 sig	0.029 sig

Above table5.6 shows significance in longer duration subjects between drivers and conductors, which indicates drivers are affected more as duration increases which means apart from noise additional vibration factor plays role as synergistic effect.

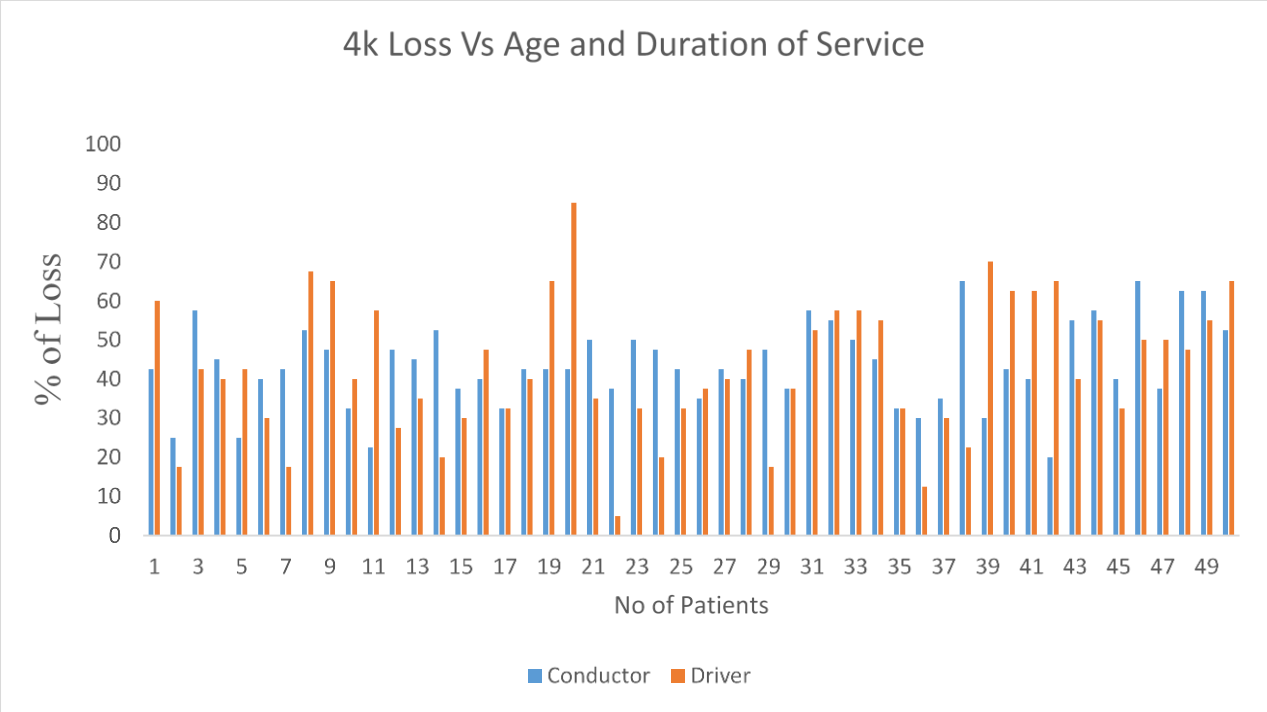


Fig 5.3 graphical presentation of hearing loss in 4 KHz against age and service.

The above graph represents more than 80% both driver and conductors are affected, but few drivers were affected worse than drivers with higher groups. This may be due to present mode of service like double and triple duty (16 and 24 hours of service with 48 hours break) or younger age group are sensitive to

noise and vibration (61,2,3,4,5) & further once they reach 75% threshold they are not sensitive to NIHL but for age related loss will be there. So we need to compare the same age and exposure groups to know the sensitivity of each age and exposure groups with board range of noise and vibration exposure.

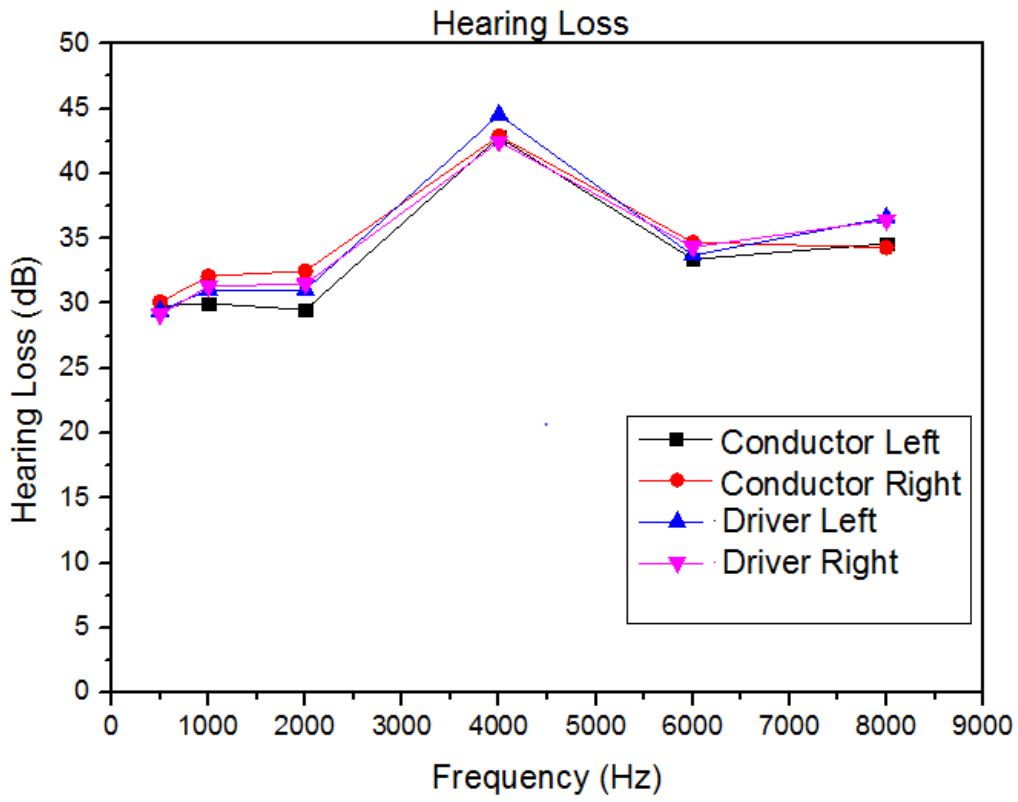


Fig 5.4

Fig 5.4 shows the 4KHZ dip (tented) and more or less similarity between right and left indicates occupational noise induced HL. Mild increased loss in left driver and right conductor with the corresponding opposite side indicates the shadow effect (62).

Discussion

SNHL being one of the worst, irreversible, health hazard faced in the occupational disease needs more attention to prevention in industrial population.

This loss is manifested as a silent killer in the view that most of the people with NIHL suffer from high frequency loss (3, 4, 5, 6,) which the subjects are unaware of it. High frequency loss is not involved in the speech reception in the beginning (3, 4, 5, 6,), but by the time they realize subject will land up in an irreversible SNHL (3, 4, 5, 6,).

But SNHL due to NIHL is a preventable disease (3, 4, 5, 6,) in case of occupational disease if we strictly abide by rules and regulations regarding prevention.

Many factors are involved in the etiology of NIH L (1, 2, and 32). Few are modifiable. Noise is the one main factor that is well known and well-studied with vast documentation since AD's. Vibration (32) one of the ergonomic condition faced by the drivers, which is also a modifiable factor, is studied indirectly in this study.

In this study, out 103 sample subjects (53 conductors and 50 drivers) 100 subjects are those who strictly fulfill study criteria. Two groups – drivers and conductors are taken for study in equal proportion. Sex factor is not considered in this study because of the type of profession is mostly handled by males in our country and all of our subjects are males only. Though sex factor plays role male are affected more than females. (ISO 7009).but Niels Christian stinker studied a controversy for this expression (64).

Age group being the most confounding factor of our study, it is considered more and distribution shown in table. As the study aims for prevention only the age group of more than 18 yrs and less than 58 yrs is considered.

According to Robinson and Sutton age related hearing loss variation are observed from the age of 18 and above, this variation is eliminated by using Robinsons and Sutton distribution curve and formula.

In our cross-sectional study, all subjects are otologically normal. Not even a single subject complained about tinnitus, one of the earliest sign of NIHL. All systemic illness were filtered out by testing blood pressure, random blood sugar and hemoglobin levels (of more than 12 mcg/dl and random sugar less than 140 mg/dl). Smoking does not involve in NIHL as a lone factor studied by Jukka Starck, Esko Toppila, Ilmari Pyykköa in 1999(10). As most of our subjects consume alcohol in random habits and in view of difficult to get pure alcohol free subjects alcohol consumption is not considered and there are no separate study results for its effect on hearing in this study.

In age distribution study, the correlation between the age and the hearing loss has been calculated as 11.5% (30-40 yrs), 12.24% (41-50 yrs), 24.42 (51-60 yrs) with p-values of 0.002 and service distribution with p-value 0.013, which is significant for the study. Two age groups are significant, which shows that extremes of age groups are affected more with significant difference between two groups. This shows that drivers of younger and elder most groups are affected with higher NIPTS even though both groups are exposed to more or less same level of noise in the same environment. Both groups are seated on either side of the bus engine approximately with equal duration of exposure i.e. More than 8 hours. More studies have documented that younger age

groups are more sensitive to noise and affected more with considerable loss by the time of 10 yrs of exposure, followed with stable loss in the middle age, and aggravated in the older age.

Table 5.2 shows non significance for MCL hearing loss because there is no major difference but minor difference of about 3 dB loss more in drivers compared to conductors. In the same way difference in the high frequency loss too around 3dB, but there is a major difference between the drivers and conductors for low frequency loss with significant p-0.050. This shows that drivers are earlier to get affected by noise in low frequency followed by HFL, due to exposure.

Table 5.3 shows significant p-value for comparison between high and low frequencies. This indicates that conductors are affected in noise pollution with NPTS. Table 5.4 shows the significant p-value for the comparison between LFL and HFL in drivers, which indicates drivers are affected more with occupational noise induced hearing loss (NPTS). There is a major difference between left and right in drivers, which is due to "shadow effect" of right ear as in studies of Adarsh Kumar, PhD,1_ N.N. Mathur, MS,2 Mathew Varghese, MS, Dinesh Mohan, PhD, J.K. Singh, MTech, and Punnet Mahajan, PhD released in AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 47:341–348 (2005).

This above result shows that apart from noise other significant factors are involved in the NIHL. Apart from noise drivers are subjected to heat and vibration. Conductors are also subjected to heat, who seat on other side of engine but provided with cushion seat same as for passengers. So vibration should be the modifying factor for above derived significant difference in NIPTS.

But experimental studies have shown that combined exposure of vibration and noise has significant effect on SNHL (NIHL), but no effect on individual exposure in animal studies.

Hearing loss using the conventional formula (that is "subject with less than or equal to 25dB loss are considered to be healthy") without eliminating the age related loss were also calculated separately for high and low frequency loss and compared for both driver and conductors using unpaired tests. When high frequency and low frequency are compared in each group there is highly significant correlation. This is because high frequency perception is affected earlier in NIHL and low frequency in the later period.

When duration of service is considered (table 5.6) there is significant difference between the two groups for low frequency loss (p-value=0.0094) and high frequency loss (0.0391). But as a whole impairment (NPTS) increases with the duration of service in subjects more than 10 years of service. Subjects with less than 10 years of service are least affected with 3% difference and higher the duration the loss is around 7%.

When this values are considered it is very negligible difference. Helmut Seidel, Barbara Harazin, Kristina Pavlas, Christine Sroka, Jörg Richter, Ralph Blüthner, Udo Erdmann, Jan Grzesik, Barbara Hinz and 1 more in 1988(8) study found significant combined effect of noise and vibration exposure, but prolonged duration varies with individual susceptibility.

Takashi Miyakita, Hajime Miura and Makoto Futatsuka in 1987(9) studied relationship between VIWF and NIHL and found that subjects with vibration syndrome disease have an increased prevalence of NIPTS.

Griffin in his article (56) for demand for the necessity of minimal health hazard safety for employees subjected to vibration exposure by European Union derived to the conclusion that neither EAV nor ELV are insufficient to identify vibration health hazard. It may be better to have other qualitative guidance.

Dozent Dr. S c. med. Helmut Seidel in their recent study in 2007(11) in American journal of industrial medicine have shown minor synergistic effect of vibration with noise in NIPTS in human study.

Criteria for the NIPTS (56), that there is no significant major difference between the right and left ear both for high and low frequency is fulfilled in our study.

Pyykkö & J. Starck in 1982 studied the pathological association between HAV syndrome relations to hearing loss and found that sympathetic hyper reaction of vascular response is not restricted to hand arm but affects the whole system by which hearing was also affected.

Pyykkö, J. Pekkarinen, J. Starck in their study (1987)(7) have shown that there is no significant risk in SNHL due to combined exposure, but observed significant risk in subjects with VWF.

Jukka Starck, Jussi Pekkarinen and Ilmari Pyykkö again in 1998 studied the impact of impulse noise and vibration effect on SNHL and came to the conclusion that subjects exposed to impulse noise experienced risk but not for hand arm vibration exposure. But in this study too subjects with VWF experienced the significant risk.

But recent study by M.J. Griffin in view of minimum health and safety requirements (33) for vibration exposure showed present limits as EAV and ELV are not sufficient

to prevent vibration syndrome and will be effective only by qualitative refinement of exposure, because the value differs state to state in European union and abroad. There is no specified limit for vibration (M.J.Griffin 2012) (2, 32). In our study measured vibration is beyond action value (0.5ms2) and limit value (9.1ms-1 for 8 hrs of exposure) that is 9.5 ms-1. Our subjects are engaged for more than 8 hrs upto 24 hrs sometimes(by method of double and triple attendance criteria) which is more harmful for hearing threshold.

Helmut Seidel, Ralph Blüthner, Janos Martin, Gerhard Menzel, Rudolf Panuska, Peter Ullsperger in 1992 studied the auditory event related brain potential in relation WBV and noise(60 and 80 dBs) with sinusoidal acceleration 2 ms-1 ,which shows significant risk in the shortening of brain potential with an increasing exposure(14). It means cumulative effect of noise and WBV on hearing impairment reducing the inhibitory action of outer hair cells and its loss.

Tatsuya Yamasobaa, b, Jochen Schachta, Fumi Shojia, c, Josef M Millera,in 1999 studied the attenuation effect of iron chelator (15) on NIHL and significant prophylactic effect. This study confirms the chelation effect of desferoxamine mesylate and glial cell line-derived neurotrophic factor (GDNF) provides attenuation of NIHL by reducing the effect of reducing oxygen species free radicals released by vascular injury due to hyper sympathetic action. This study also proves the mechanism of NIHL is similar to VWF syndrome, which affects also hearing. Theoretically it means synergistic effect will be present in combined exposure.

However most of the studies supports the mild synergistic effect, there was contradictory effect studied by the Group of acoustics and vibrations in human GRAVI

in 2006 (izhumi) (61) found antagonistic effect at4kHz.this as supported by Griffins (1996) (2) by Coles et al study. He supported the improvement in hearing after combined exposure. But the majority of papers supports the synergistic effect of noise and vibration on hearing loss.

In this study overall comparison between driver and conductor shows non-significant result (p-value-0.520), but minor significant difference of 3% loss more, is to be considered as synergistic effect in view of mechanism by which vibration and noise affects hearing. Drivers are not able to attenuate noise in view of their profession who needs regular contact with surroundings to avoid accidents, So it is must to protect this professionals (driver) from hearing loss by organizing and implementing noise and vibration management program, which also helps in documentation and refinement in automobile engineering that will be helpful for public in view of safety and health hazards.

NIHL affects only 4 KHz (1, 2, 3, 4, 5, and 56) (table 3.2)) in the earlier period, which does not influence speech reception. Patient will not turn up for consultation. But in due course of years it affects speech frequency. So it is mandatory to perform Audio logical evaluation to identify risk subjects, so that they can be transferred to safe job before it affects speech perception.

Though most (80%) of the subjects are affected in 4 kHz the reason for individual susceptibility with 0% loss is not understood. (61)

Drivers and conductors may be subjected to regular medical checkup to analyze their hearing by PTA cross checked with DPOAE, which is the confirmatory test for NIHL.

Risk groups should be analyzed and may be treated with free radical chelating agent as prophylaxis (15). Since most of the buses observed were not upto the standards as per OSHA (59) and ISO standards (58). Noise and vibration control program to be implemented and strictly followed for future documentation.

Following matters to be considered while implementation of "The noise and vibration controlled program" (33)

- Regular medical health checkup (PTA and OAE) and documentation while appointment and there after yearly.
- Limitations and duration of exposure to be strictly followed (not to allow for double and triple attendance that is not provisions as per OSHA and ISO 5349 (1986),
- Risk assessment and mobilization of subject to safe zone.
- Knowledge program and training on risk establishment and necessity of program in preservation of vital sense.
- Provision of auxiliary equipment that reduces the risk (seat refinement and handles)
- Appropriate driver cabin with refined ergonomics that reduces vibration other mode of stress.
- Maintenance program for reducing vibration and noise of vehicle (regular condemnation of aged vehicle).
- Maintenance of roads up to standards is important section of program.

Most of the study are experimental, where the acceleration with various frequency combinations are limited are not enough to get vast knowledge about the combined

effect of WBV and NOISE. Due to scarcity of studies and the importance of prevalence (combined exposure), there is a need to explore new method of large scale case controlled study in relation to various factor that affects NIHL.

Conclusion

It is concluded that the hearing impairment experienced by driver is due to occupational hazard and synergism of noise of and whole body vibration is significantly significant with only 3%. Further we need large scale case controlled study with different methodology for better knowledge of combined effect of WBV and noise on hearing using DPOAE.

Vibration is a mode by which noise is generated, so without vibration there cannot be noise in automobiles. Vibration is the basic thing that affects drivers in NIHL, who is in continuous contact with engine control that needs to be refined. Noise induced hearing loss one of the major occupational disease needs more attention for the prevention, which has no treatment. Since drivers need good contact with surroundings to reach destination safely, they are not able to attenuate noise by hearing protection device. Drivers are affected physically and psychologically due to noise and WBV, which can affects their performance. So it is mandatory to provide them better ergonomics to prevent NIHL.

Organization and implementation of noise and vibration control program should be strictly followed to prevent NIHL. Better ergonomics not only helps drivers but also reduces public health hazard by reducing road traffic accidents.

So automobile industry need to be refined well to prevent occupational NIHL in drivers and conductors, where vibration also plays significant role.

Summary

- NIHL is the second most common hearing loss in whole, which is preventable but not treatable. Worldwide 16% and in India varies from 7 to 21% in different sub regions.
- Drivers and conductors are one of the sub groups exposed to noise, who are unable to attenuate, so can avoided only by modifying different factors that involves in NIHL.
- Hearing loss one of minor sign in Vibration syndrome, which affects drivers performance in many ways physically and psychologically that burdens public safety.
- NIHL clinically does not present in the earlier stage.by the time patient comes to consult they would have reached NIPTS, which is non treatable.
- But still worldwide lacks a clear study for synergism of vibration with noise on SNHL, few antagonistic result are also faced as vibration improves TTS.
- So in this study we choose combined effect of vibration and noise faced by drivers over conductors, who are not subjected to vibration but subjects to noise similar to drivers.
- Otologically normal 103 subjects were selected, all subject fulfills our selection criteria.
- All subjects were tested for hemoglobin, random blood sugar, blood pressure measurement, tuning fork test and pure tone audiogram.

- Age related factor as eliminated by using Robinsons and Suttons formula and web based calculator for occupational hearing impairment.
- All data's were analyzed using chi-square, t-test and paired t-est.
- Drivers and conductors were compared for HFL, LFL and total loss in relation to age and service.
- Out of 103 subjects 14 were normal (subject with 25 dBs and less are considered 0 % loss).
- One driver was measured as profound loss with 90dB.
- No patients were complained for tinnitus.
- Noise level measured randomly in 4 yrs. old bus as 75-95 dBs
- Vibration in driver seat as x-axis 0.5 ms-2, y-5 to 9.5 ms-2, z-0.8 ms-2
- Overall drivers suffered from occupational hearing loss with 3dB more than conductors, which is very mild significance.
- Both drivers and conductors are affected with HFL, but overall drivers are affected with 10 dB more in average.
- Drivers are significantly affected in LFL in addition to HFL in comparison to conductors, which indicates severity of NIHL after long duration of exposure.
- When right and left ear are compared there is no significant difference for attenuation.
- Both subjects had significant loss for HFL ($p= 0.000<0.05$) in comparison with speech frequency, but drivers are affected with 10 dB greater than conductor.
- So in conclusion drivers does has additional loss, which is considered to be due to vibration. It is also concluded that variable susceptibility noise and vibration

were observed that may the habit of food and physical activity that involves metabolic clearance of free radicles.

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ANNEXURES

ANNEXUER 1: ROBINSON AND SUTTON'S REFERENCE TABLE.

Table III. Values of the dispersion parameters a′ ($\times 10^5$) and b′

Frequency kHz	a′ × 10⁵, dB/year²				Median	b′, dB				Median
	males					males				
	01	05	08			01	05	08		
0.125	–	52	13		38	–	7.4	5.8		6.6
0.25	121	55	93		93	7.6	6.8	5.0		6.8
0.5	77	149	155		149	7.8	5.7	4.8		5.7
1	281	190	82		190	5.7	5.6	5.3		5.6
1.5	280	–	–		280	7.9	–	–		7.9
2	380	410	77		380	8.6	7.3	6.1		7.3
3	447	593	159		447	11.8	6.9	6.8		6.9
4	307	611	368		368	14.9	7.4	5.8		7.4
6	306	480	331		331	16.7	8.4	8.4		8.4
8	313	707	756		707	16.6	8.6	9.6		9.6
10	–	–	–		–	–	–	–		–
12	–	–	1 609		1 609	–	–	12.0		12.0
	females					females				
	02	06	07	09		02	06	07	09	
0.125	–	82	41	10		–	7.1	4.7	5.8	5.8
0.25	274	111	39	93	102	6.2	6.9	4.1	5.0	5.6
0.5	221	88	19	153	125	7.7	6.9	3.9	4.8	5.9
1	368	36	58	82	71	6.0	7.2	3.6	5.3	5.7
1.5	334	–	–	–	334	6.3	–	–	–	6.4
2	347	168	178	162	173	6.5	6.8	3.7	6.0	6.3
3	337	260	248	904	301	7.1	6.3	3.6	6.5	6.4
4	447	367	346	986	409	6.9	6.6	4.4	8.2	6.8
6	404	436	555	724	499	9.3	8.0	4.6	8.9	8.5
8	524	487	709	1 231	506	10.0	10.0	4.7	8.9	9.5
10	–	–	–	–	–	–	–	–	–	–
12	–	–	–	1 616	1 616	–	–	–	11.9	11.9

MASTER CHART 1: CALCULATED LOSS from chart 2.

S. No	D/C	Low Frequency			High Frequency			E. Age Loss	MCL OCC	AGE	Service
		Left	Right	Total	Left	Right	Total				
001	C	7.5	5	5.4	7.5	17.5	9.2	57.60	10.00	35	8
002	C	0	0	0.0	0	0	0.0	40.80	0.00	36	6
003	C	0	0	0.0	10	0	1.7	39.02	1.56	37	8
004	C	5	5	5.0	12.5	12.5	12.5	35.68	5.63	38	8
005	C	0	0	0.0	0	0	0.0	51.72	0.00	38	9
006	C	0	0	0.0	0	7.5	1.3	42.69	0.63	38	9
007	C	17.5	5	7.1	35	40	35.8	33.82	15.00	39	9
008	C	0	0	0.0	17.5	20	17.9	13.39	10.00	39	10
009	C	7.5	10	7.9	20	22.5	20.4	12.13	11.09	39	10
010	C	5	5	5.0	12.5	12.5	12.5	31.18	5.63	39	12
011	C	17.5	5	7.1	2.5	15	4.6	54.69	10.63	40	12
012	C	7.5	32.5	11.7	10	12.5	10.4	13.41	15.00	42	12
013	C	0	0	0.0	2.5	2.5	2.5	14.51	0.31	43	12
014	C	15	7.5	8.8	40	65	44.2	36.08	30.31	43	12
015	C	0	0	0.0	10	0	1.7	50.55	0.94	45	12
016	C	0	27.5	4.6	10	17.5	11.3	14.52	11.88	45	13
017	C	5	0	0.8	15	15	15.0	23.87	2.19	45	13
018	C	0	0	0.0	0	12.5	2.1	22.12	0.31	45	14
019	C	0	2.5	0.4	17.5	12.5	13.3	15.84	24.69	45	14
020	C	20	15	15.8	20	35	22.5	47.32	30.31	46	15
021	C	32.5	7.5	11.7	17.5	22.5	18.3	16.69	21.25	48	15
022	C	5	0	0.8	7.5	0	1.3	29.57	3.75	48	19
023	C	17.5	15	15.4	22.5	20	20.4	33.53	15.00	48	19
024	C	7.5	2.5	3.3	20	5	7.5	45.93	1.25	49	20
025	C	45	25	28.3	45	12.5	17.9	33.80	8.75	49	20
026	C	2.5	5	2.9	7.5	17.5	9.2	35.52	2.81	50	20
027	C	7.5	22.5	10.0	17.5	17.5	17.5	31.50	12.50	50	24
028	C	5	10	5.8	17.5	12.5	13.3	21.31	8.75	52	25
029	C	-2.5	0	-2.1	15	12.5	12.9	26.20	10.31	52	25
030	C	32.5	5	9.6	30	20	21.7	24.70	11.88	52	26
031	C	12.5	5	6.3	42.5	27.5	30.0	31.27	19.06	53	26
032	C	12.5	5	6.3	42.5	27.5	30.0	28.86	7.64	54	28
033	C	7.5	10	7.9	55	50	50.8	29.88	24.69	54	28
034	C	0	2.5	0.4	30	0	5.0	0.00	5.00	54	28
035	C	10	17.5	11.3	0	7.5	1.3	26.99	10.94	55	28
036	C	7.5	10	7.9	25	12.5	14.6	17.78	20.94	55	28
037	C	0	0	0.0	30	7.5	11.3	8.78	3.75	55	28
038	C	2.5	62.5	12.5	27.5	70	34.6	37.69	13.44	55	28
039	C	2.5	12.5	4.2	40	0	6.7	36.34	42.50	55	28
040	C	10	15	10.8	17.5	15	15.4	21.10	16.88	55	28
041	C	0	2.5	0.4	17.5	12.5	13.3	30.67	34.06	55	28

042	C	22.5	22.5	22.5	42.5	2.5	9.2	30.08	15.94	56	28
043	C	7.5	0	1.3	32.5	42.5	34.2	16.92	20.94	56	28
044	C	10	0	1.7	0	0	0.0	13.31	0.63	56	29
045	C	5	5	5.0	15	32.5	17.9	27.62	6.56	56	29
046	C	2.5	2.5	2.5	30	27.5	27.9	22.75	24.69	56	29
047	C	17.5	10	11.3	5	10	5.8	30.08	8.44	56	29
048	C	2.5	10	3.8	7.5	25	10.4	44.02	17.81	56	30
049	C	0	2.5	0.4	12.5	40	17.1	14.77	2.19	57	30
050	C	25	22.5	22.9	45	42.5	42.9	14.50	37.81	58	33

S. No	D/C	Low Frequency			High Frequency			E. Age Loss	MCL OCC	Age	Service
		Left	Right	Total	Left	Right	Total				
001	D	12.5	32.5	15.83	32.5	37.5	33.33	25.46	2.66	32	8
002	D	0	0.0	0.00	0.0	0.0	0.00	0.00	0.00	32	8
003	D	0	7.5	1.25	7.5	7.5	7.50	9.61	1.25	35	8
004	D	0	2.5	0.42	2.5	7.5	3.33	4.56	0.94	35	8
005	D	5	15.0	6.67	15.0	12.5	12.92	10.76	0.94	35	8
006	D	0	0.0	0.00	0.0	0.0	0.00	0.00	0.00	35	8
007	D	0	0.0	0.00	0.0	0.0	0.00	5.40	0.00	35	8
008	D	40	57.5	42.92	57.5	37.5	40.83	6.43	35.31	36	9
009	D	7.5	35.0	12.08	35.0	32.5	32.92	5.94	41.25	36	9
010	D	10	10.0	10.00	10.0	7.5	7.92	9.66	8.44	36	9
011	D	0	47.5	7.92	47.5	55.0	48.75	10.19	39.69	36	9
012	D	5	5.0	5.00	5.0	0.0	0.83	14.89	0.63	37	9
013	D	2.5	0.0	0.42	0.0	0.0	0.00	15.53	0.00	38	9
014	D	2.5	0.0	0.42	0.0	0.0	0.00	17.94	0.00	38	10
015	D	10	0.0	1.67	0.0	0.0	0.00	0.00	0.00	38	11
016	D	17.5	30.0	19.58	30.0	12.5	15.42	12.82	15.31	38	12
017	D	5	12.5	6.25	12.5	7.5	8.33	11.99	12.19	39	12
018	D	0	25.0	4.17	25.0	5.0	8.33	0.00	4.69	39	13
019	D	17.5	52.5	23.33	52.5	62.5	54.17	14.46	36.25	39	13
020	D	17.5	90.0	29.58	90.0	107.5	92.92	20.15	43.44	40	13
021	D	2.5	0.0	0.42	0.0	0.0	0.00	20.36	0.00	41	14
022	D	0	0.0	0.00	0.0	0.0	0.00	12.28	0.00	41	14
023	D	0	0.0	0.00	0.0	0.0	0.00	0.00	0.00	41	14
024	D	5	0.0	0.83	0.0	0.0	0.00	20.62	0.00	42	15
025	D	0	0.0	0.00	0.0	22.5	3.75	15.87	4.69	43	15
026	D	0	0.0	0.00	0.0	0.0	0.00	34.71	0.00	45	15
027	D	12.5	27.5	15.00	27.5	20.0	21.25	21.58	15.63	45	15
028	D	0	17.5	2.92	17.5	25.0	18.75	22.36	13.13	46	16
029	D	15	17.5	15.42	17.5	0.0	2.92	26.84	6.25	46	16
030	D	2.5	17.5	5.00	17.5	17.5	17.50	28.27	13.31	47	16
031	D	22.5	20.0	20.42	20.0	25.0	20.83	23.99	28.75	47	17
032	D	12.5	37.5	16.67	37.5	45.0	38.75	27.75	25.63	47	17

033	D	20	55.0	25.83	55.0	52.5	52.92	18.33	28.75	48	17
034	D	5	35.0	10.00	35.0	32.5	32.92	19.82	24.69	48	18
035	D	0	5.0	0.83	5.0	22.5	7.92	34.65	6.25	48	18
036	D	0	0.0	0.00	0.0	0.0	0.00	31.50	6.25	49	18
037	D	20	10.0	11.67	10.0	17.5	11.25	0.00	18.44	49	18
038	D	20	0.0	3.33	0.0	0.0	0.00	24.80	0.00	49	19
039	D	25	52.5	29.58	52.5	40.0	42.08	0.00	39.69	49	20
040	D	22.5	37.5	25.00	37.5	60.0	41.25	27.92	36.56	50	20
041	D	7.5	30.0	11.25	30.0	32.5	30.42	29.21	13.31	50	21
042	D	7.5	32.5	11.67	32.5	42.5	34.17	25.37	14.69	50	21
043	D	0	2.5	0.42	2.5	0.0	0.42	28.85	1.56	50	21
044	D	12.5	52.5	19.17	52.5	37.5	40.00	23.42	20.00	51	21
045	D	0	20.0	3.33	20.0	0.0	3.33	27.34	1.88	52	23
046	D	0	17.5	2.92	17.5	12.5	13.33	28.62	15.94	52	25
047	D	5	22.5	7.92	22.5	12.5	14.17	22.53	12.19	52	25
048	D	35	40.0	35.83	40.0	65.0	44.17	31.23	30.63	53	27
049	D	45	50.0	45.83	50.0	55.0	50.83	25.97	45.00	54	28
050	D	7.5	32.5	11.67	32.5	42.5	34.17	22.68	20.94	56	28

AGE LOSS: Expected age loss for the corresponding age calculated from Robinsons & Sutton's formula using web based master calculator.

MCL OCC: Master calculator loss due to occupation after eliminating age related factor by NIOSH 1998 formula using web based calculator.

LFL: calculated from values 500 Hz, 1 KHz and 2 KHz by conventional formula.

HFL: calculated from values of 4 KHz, 6 KHz and 8 KHz by conventional formula.

Conventional formula for right or left: (a +b +c /3) * 1.5, for total loss % =5x+y/6.

D: Drivers.

C: Conductor.

MASTERCHART 2: RECORDED PTA DATA FROM PATIENTS.

S. No	D/C	Age	Left Ear						Right Ear					
			Low Frequency			High Frequency			**Low Frequency**			**High Frequency**		
			500	**1K**	**2K**	**4K**	**6K**	**8K**	**500**	**1K**	**2K**	**4K**	**6K**	**8K**
001	D	32	35	30	35	60	45	35	25	35	40	60	50	40
002	D	32	20	15	10	15	5	25	25	10	25	20	10	20
003	D	35	20	10	25	45	20	25	15	30	30	40	30	20
004	D	35	30	15	25	35	20	25	15	5	20	45	25	20
005	D	35	25	35	25	55	35	15	25	25	25	30	35	35
006	D	35	20	10	25	40	5	25	20	30	15	20	0	20
007	D	35	40	20	10	30	10	0	15	25	10	5	15	0
008	D	36	60	55	40	70	70	50	50	35	40	65	50	35
009	D	36	30	25	35	60	35	50	35	45	55	70	40	30
010	D	36	30	35	30	40	30	25	25	25	25	40	30	20
011	D	36	30	20	15	55	70	45	35	45	35	60	70	55
012	D	37	35	25	25	35	35	15	25	20	30	20	30	15
013	D	38	20	20	40	35	25	15	20	10	30	35	20	20
014	D	38	30	20	30	5	5	15	30	25	35	35	20	20
015	D	38	35	25	35	30	5	15	20	30	40	30	5	25
016	D	38	30	40	40	60	35	40	35	30	35	35	35	30
017	D	39	25	30	30	30	35	35	30	35	35	35	30	25
018	D	39	20	25	10	45	45	35	15	15	5	35	25	25
019	D	39	30	35	45	70	60	50	20	35	45	60	65	75
020	D	40	45	30	35	80	100	75	55	40	50	90	100	100
021	D	41	20	35	25	25	0	15	30	45	25	45	5	20
022	D	41	15	30	25	0	10	25	30	40	25	10	25	30
023	D	41	30	15	5	40	0	20	25	10	20	25	0	20
024	D	42	25	30	30	20	30	20	20	20	20	20	25	25
025	D	43	30	15	25	30	5	25	40	50	40	35	50	35
026	D	45	15	5	20	40	20	10	20	15	25	35	15	5
027	D	45	35	30	35	40	55	35	35	45	30	40	40	35
028	D	46	15	35	25	45	35	30	20	25	35	50	35	40
029	D	46	45	35	25	5	60	45	45	45	30	30	5	35
030	D	47	20	25	35	40	35	35	25	35	30	35	35	40
031	D	47	35	50	35	50	40	25	35	45	35	55	40	30
032	D	47	35	40	25	60	40	50	50	40	50	55	60	50
033	D	48	45	40	30	60	65	60	45	40	30	55	65	60
034	D	48	20	30	35	50	55	40	30	45	50	60	45	35
035	D	48	20	25	30	30	25	30	20	20	25	35	40	45
036	D	49	20	5	20	10	0	10	20	10	25	15	5	15
037	D	49	35	45	35	25	40	30	35	50	40	35	50	25
038	D	49	35	45	35	25	10	35	50	40	25	20	5	30
039	D	49	45	35	45	70	55	55	30	40	55	70	50	35
040	D	50	40	45	35	60	50	40	35	45	50	65	70	60

041	D	50	25	30	35	65	20	50	35	20	30	60	35	45
042	D	50	25	30	35	65	20	55	20	25	30	65	45	50
043	D	50	20	30	15	45	10	25	35	20	30	35	25	15
044	D	51	30	35	35	50	60	70	40	50	40	60	40	50
045	D	52	30	20	25	55	30	30	25	35	15	10	20	20
046	D	52	25	25	25	50	35	25	25	20	20	50	25	25
047	D	52	20	25	40	55	35	30	25	30	35	45	25	30
048	D	53	50	55	40	25	60	70	50	60	50	70	65	70
049	D	54	50	65	50	45	60	70	55	65	55	65	60	60
050	D	56	25	30	35	65	20	55	20	25	30	65	45	50

			Left Ear						Right Ear					
S. No	D/C	**Age**	Low Frequency			High Frequency			**Low Frequency**			**High Frequency**		
			500	**1K**	**2K**	**4K**	**6K**	**8K**	**500**	**1K**	**2K**	**4K**	**6K**	**8K**
001	C	35	30	35	25	30	40	20	30	30	25	55	25	30
002	C	36	25	20	10	20	10	10	20	25	30	25	10	20
003	C	37	30	20	0	40	35	20	20	30	20	55	15	5
004	C	38	30	30	25	55	20	25	30	35	20	25	40	35
005	C	38	20	20	15	25	40	10	20	20	10	25	15	25
006	C	38	25	15	25	10	35	25	30	30	15	35	25	30
007	C	39	35	30	45	45	55	45	25	30	30	35	60	60
008	C	39	20	25	20	50	35	25	15	25	20	50	45	20
009	C	39	40	30	20	50	35	30	45	25	25	50	40	30
010	C	39	30	30	25	55	20	25	30	35	20	25	40	35
011	C	40	35	35	40	35	20	25	30	25	30	35	40	30
012	C	42	30	30	30	40	35	20	50	45	45	45	35	20
013	C	43	20	20	20	35	25	20	15	15	20	40	25	15
014	C	43	30	35	40	60	45	50	25	30	35	70	55	80
015	C	45	20	25	20	40	25	30	30	20	15	25	25	20
016	C	45	25	25	25	40	30	25	50	50	30	45	40	25
017	C	45	30	25	30	40	45	20	20	20	10	30	40	35
018	C	45	20	20	20	25	20	15	15	15	20	45	35	20
019	C	45	20	25	20	45	40	25	20	25	35	45	35	20
020	C	46	35	40	40	50	30	35	30	35	40	70	45	30
021	C	48	55	50	35	40	35	35	30	30	30	50	40	30
022	C	48	30	30	25	40	20	30	25	20	30	30	20	25
023	C	48	35	35	40	50	35	35	35	30	40	30	40	45
024	C	49	30	35	25	45	45	25	30	30	20	25	35	25
025	C	49	50	55	60	70	45	50	40	45	40	30	30	40
026	C	50	25	25	30	25	30	35	25	25	35	30	35	45
027	C	51	30	25	35	45	35	30	50	40	30	45	35	30

028	C	52	30	25	30	35	30	45	20	40	35	30	25	45
029	C	52	20	25	25	70	15	40	20	25	25	55	15	30
030	C	52	50	50	40	55	40	40	30	25	30	35	35	45
031	C	53	30	30	40	60	50	50	30	25	30	50	40	40
032	C	54	30	30	40	50	60	50	30	25	30	50	40	40
033	C	54	30	25	35	60	55	70	25	30	40	55	50	70
034	C	54	20	20	20	40	35	60	25	30	25	40	30	5
035	C	55	25	30	40	25	10	40	30	40	40	35	15	40
036	C	55	25	30	35	50	35	40	25	30	40	35	30	35
037	C	55	30	25	20	50	45	40	25	20	25	30	25	35
038	C	55	25	30	25	40	40	50	60	70	70	65	70	80
039	C	55	25	30	25	65	50	40	35	25	40	30	10	35
040	C	55	25	40	30	35	25	50	30	45	30	35	20	50
041	C	55	20	25	20	45	40	25	20	25	35	45	35	20
042	C	56	30	40	50	60	50	50	30	45	45	15	25	40
043	C	56	25	30	35	65	20	55	20	25	30	65	45	50
044	C	56	25	30	40	10	0	25	20	15	35	30	5	10
045	C	56	25	30	30	15	50	40	25	35	25	35	50	55
046	C	56	25	25	30	70	20	45	25	25	30	70	20	40
047	C	56	35	30	45	30	20	35	30	40	25	30	25	40
048	C	56	25	30	25	50	25	15	30	25	40	55	35	35
049	C	57	25	20	25	30	30	40	25	25	30	45	50	60
050	C	58	30	40	55	45	55	65	30	40	50	65	50	45

PROFORMA

Name…….. **Age**……

Duration of service……

Type of job ◯ Driver ◯ Conductor ◯ Driver cum conductor

Duration of daily driving/travel ◯ 1-10 ◯ 10-20 ◯ 20-30 ◯ 30-40

Do u use horn frequently ◯ Yes ◯ No

Type of bus engine; ◯ Front ◯ Rear

Do u feel vibrations during driving ◯ Yes ◯ No

Daily driving/travel distance Kms ◯ 200 – 400 ◯ 400-600 ◯ 600-800

Chief complaints;

Present History 1) Fullness in ear ◯ Yes ◯ No

2) Tinnitus ◯ Yes ◯ No

3) Giddiness, dizziness ◯ Yes ◯ No

4) Noise intolerance ◯ Yes ◯ No

5) Ear pain ◯ Yes ◯ No

6) Ear discharge ◯ Yes ◯ No

7) Numbness in foot or fingers ◯ Yes ◯ No

8) Pain in finger on exposure to cold ◯ Yes ◯ No

Past illness history; DM2 / HTN /ear discharge/ear drops use/ear surgery/frequent self-medications / PT /head injury/

Personal habits 1) Alcohol ◯ Yes ◯ No

2) Smoking ◯ Yes ◯ No

3) Pawn chewing ◯ Yes ◯ No

4) Drug abuse ◯ Yes ◯ No

5) Medications ◯ Yes ◯ No (if Yes specify)

Family history of hearing defects;

Hearing status before joining to job

Local examination; **Right** **Left**

Pinna

EAC :

Tympanic membrane:

Mastoid :

Facial nerve :

Tuning fork test

Rinnes :

Webers :

ABC :

Total hearing impairment due to NIHL (MCL):

Total impairment by WHO formula:

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LB SR 011 Audiolab Control Report

Audiometer Calibration Certificate

Aud. Mfr.:	LABAT	Earphone type:	DD 45 ☒ / TDH 50 ☐
Aud. Model:	AudioLab	Earphone s/n (R/L):	WT125235 - WT125225
Aud. s/n:	AUL 14085	Bone conductor type:	RADIOEAR B-71
Customer:		Bone s/n:	A32745
Location:			

Calibrated by: **LABAT s.r.l.** Via Roma 14/2 - 30037 Scorzè (VE) Italy
Tel. +39 041 457299 Fax +39 041 4574940 e-mail: info@labat.it

Catena di misura utilizzata:

Descrizione	Costruttore	Modello	S/n	Certificato	Scadenza certificato
Fonometro	B&K	2260	2168577	09-1705-FON	02/03/2015
Microfono	B&K	4189	2146181	09-1705-FON	02/03/2015
Mastoide artificiale	B&K	4930	2022850	1159310	04/11/2015

Date: 19-11-2014 Technician: Roberto Zanon

70 dB HL Hearing Level and Frequency Test

Freq. Hz	Freq. Dev. %	Freq. measure	Test result	Level SPL DD 45	Level SPL TDH 50	Microphone correction	Level Dev. dB	R Level measure	Test result	L Level measure	Test result
125	+/- 3 %	125	✓	117,5	117.5	0	+/- 3	118,0	✓	117,7	✓
250	+/- 3 %	250	✓	97,0	96.5	0	+/- 3	97,0	✓	96,7	✓
500	+/- 3 %	500	✓	83,0	83.5	0	+/- 3	83,4	✓	83,1	✓
750	+/- 3 %	750	✓	76,5	78.5	0	+/- 3	76,1	✓	76,7	✓
1000	+/- 3 %	1000	✓	76,0	77.5	-0.13	+/- 3	76,5	✓	76,1	✓
1500	+/- 3 %	1500	✓	78,0	77.5	0	+/- 3	78,0	✓	77,7	✓
2000	+/- 3 %	2000	✓	78,0	81.0	-0.47	+/- 3	77,8	✓	77,5	✓
3000	+/- 3 %	3000	✓	78,0	79.5	0	+/- 3	78,2	✓	78,0	✓
4000	+/- 3 %	4000	✓	79,0	80.5	-1.59	+/- 3	79,5	✓	79,6	✓
6000	+/- 3 %	6000	✓	90,5	83.5	-2.82	+/- 5	90,5	✓	90,8	✓
8000	+/- 3 %	8000	✓	82,0	83.0	-4.05	+/- 5	81,9	✓	82,1	✓

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LB SR 011 Audiolab Control Report

Bone Vibrator Test

Freq. Hz	Test dB HL	Level SPL	Deviation dB	Level measure	Test result
250	25	73.3	+/- 3	73,5	✓
500	45	84.5	+/- 3	84,8	✓
750	45	75.3	+/- 3	75,3	✓
1000	45	69.7	+/- 3	70,1	✓
1500	45	64.5	+/- 3	66,9	✓
2000	45	59.9	+/- 3	59,9	✓
3000	45	58.3	+/- 3	58,8	✓
4000	45	59.4	+/- 3	59,5	✓
6000	45	58.1	+/- 3	58,3	✓

High Frequency - Hearing Level and Frequency Test

Earphone type: _____

Freq. Hz	Freq. Dev. %	Freq. measure	Test result	Level HL	Level SPL	R Level measure	Test result	L Level measure	Test result
8000	+/- 3 %			70	86.0				
9000	+/- 3 %			70	87.0				
10000	+/- 3 %			70	91.5				
11200	+/- 3 %			70	85.0				
12500	+/- 3 %			60	87.5				
14000	+/- 3 %			45	75.0				
16000	+/- 3 %			40	82.0				
18000	+/- 3 %			40	N.S.				
20000	+/- 3 %			20	N.S.				

Note

Audiometro tarato in conformità alle norme ISO 389 ed ANSI S3.6

Signature: _____